A Dissertation on

STUDY ON DIAGNOSTIC VALUE OF SPOT PROTEIN CREATININE RATIO WITH 24 HOURS URINE PROTEIN RATIO IN ANTENATAL WOMEN WITH PREECLAMPSIA

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL

UNIVERSITY CHENNAI.

in partial fulfilment of the regulations
for the Award of the degree of

M.S., (Obstetrics & Gynaecology)

Branch – II

INSTITUTE OF SOCIAL OBSTETRICS AND GOVT. KASTURBA GANDHI HOSPITAL FOR WOMEN AND CHILDREN

MADRAS MEDICAL COLLEGE
CHENNAI.

APRIL 2015

BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr.A.CHITHRA** on **"STUDY ON DIAGNOSTIC VALUE OF SPOT PROTEIN CREATININE RATIO WITH 24 HOURS URINE PROTEIN RATIO IN ANTENATAL WOMEN WITH PREECLAMPSIA"** during her M.S., (Obstetrics & Gynaecology) course from April 2012 to April 2015 at the Institute of Social Obstetrics, Government Kasturba Gandhi Hospital for Women and Children, Madras Medical College, Chennai.

Prof.Dr.S.Baby Vasumathi, M.D., D.G.O.,
Director,
Professor and Chief,
Institute of Social Obstertrics,
Govt. Kasturba Gandhi Hospital for
Women and Children,
Madras Medical College,
Chennai – 600 005.

Prof.Dr.S.Baby Vasumathi, M.D., D.G.O.,
Director,
Professor and Chief,
Institute of Social Obstertrics,
Govt. Kasturba Gandhi Hospital for
Women and Children,
Madras Medical College,
Chennai – 600 005.

Co-guide
Prof. Dr. Jagadeeswari,
Dept.of Pediatrics
Professor and Chief,
Institute of Social Obstertrics,
Govt. Kasturba Gandhi Hospital for
Women and Children,
Madras Medical College,
Chennai – 600 005.

Prof. Dr. R. VIMALA, M.D,
DEAN,
Madras Medical College &
Rajiv Gandhi Government General Hospital,
Chennai.

DECLARATION

I solemnly declare that the dissertation titled **"STUDY ON DIAGNOSTIC VALUE OF SPOT PROTEIN CREATININE RATIO WITH 24 HOURS URINE PROTEIN RATIO IN ANTENATAL WOMEN WITH PREECLAMPSIA"** is done by me at **INSTITUTE OF SOCIAL OBSTETRICS AND GOVT. KASTURBA GANDHI HOSPITAL FOR WOMEN AND CHILDREN,** Madras Medical College under the guidance and supervision of **Prof. Dr. Baby Vasumathi, M.D., D.G.O.,** Professor of Obstetrics and Gynaecology, Madras Medical College, Chennai.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of requirements for the award of M.S. Degree (Branch II) in Obstetrics and Gynaecology.

Place : Chennai

Date :

Dr.A.CHITHRA

ACKNOWLEDGEMENT

I Thank Dr. R. Vimala, M.D., Dean Madras Medical College for permitting me to conduct this study in Institute of Social Obstetrics and Government Kasturba Gandhi Hospital for Women and Children, Chennai.

I owe my sincere thanks to **Prof. Dr.S.Baby Vasumathi,** M.D. D.G.O. Director, ISO – KGH for her valuable guidance, during the study.

I express my profound gratitude to my guide **Prof.Dr.S.Baby Vasumathi,** M.D. D.G.O. Professor of Obstetrics and Gynecology for her unwavering support and encouragement.

My gratitude to my Assistant Professors, Statistician Mr.Ravanan my colleagues and Hospital Staff and patients for enabling me to complete the study.

CONTENTS

INTRODUCTION

According to the World Health Organisation hypertensive disease during pregnancy is a major cause of maternal and perinatal mortality and morbidity. Preeclampsia occurring in 3% to 8% of pregnancy is a major cause of maternal mortality, and it accounts for about 15% to 20% of iatrogenic preterm birth, Intrauterine growth retardation and perinatal mortality. In pregnancy ,preeclampsia is characterised by varying degrees of dysfunction of placenta and maternal response that shows features of systemic inflammation. Most consider hypertension and proteinuria to be the important hallmark of preeclampsia. As the severity of the proteinuria increases, there is also an equal increasing risk of maternal and fetal outcome as observed by Brown et al in 1995. So the principle objective in managing the preeclampsia is to predict proteinuria. Many of the Obstetrician depend up on the 24 hour urine collection method for estimating the proteinuria. Since collection of 24 hour urine proteinuria is a time consuming procedure and it is a cumbersome procedure for both the patient and also for the person handing the collection of urine. It is subjected to error because of inappropriate timing and incompleteness in its collection method. The other test that estimates the quantitative evaluation of proteinuria accurately, is the measurement of protein creatinine ratio in the spot urine as a substitute of 24 hour urine protein

excretion in quantifying proteinuria as shown by many investigators. But, there are conflicting results and there is variation in the cut off values between these studies.

This present study was done to know the efficacy of the spot urine protein creatinine ratio in assessing the severity of preeclampsia. Spot urine protein creatinine ratio method is a very easy procedure in assessing the proteinuria. It is a simple procedure. It is as effective as 24 hour urine protein ratio.

REVIEW OF LITERATURE

Preeclampsia remains main etiology of maternal and fetal mortality and morbidity worldwide. Hypertension and significant proteinuria is one of the prerequisites for the diagnosis of preeclampsia. Persistent increased proteinuria diagnoses the damage of renal system . In 1843 the obstetrician John Lever identified and separated the proteinuria in the urine of pregnant women in who had developed hypertension from that of Morbus Brightii also called as Bright's disease. After this discovery, proteinuria has been included and used to define preeclampsia and also used to classify the disease severity

Waugh et al 2005 in their studies have suggested that proteinuria can be correlated better with the severity of the disease and with other clinical measurement as it can precisely reflect the glomerulus dysfunction associated with glomerular endotheliosis of preeclampsia. Proteinuria above a threshold of $\geq$ 300 mg/day in 24 hour urine is being used as a differentiating criteria to distinguish, preeclampsia from gestational hypertension in the system of classification for the disease of hypertensive disorder of pregnancy (Davy and Macgilliary 1998). It is used both as a marker of severity and disease progression .If the

threshold of proteinuria is >5000mg in 24 hour, then that would be more predictive of adverse outcome.

In a pregnancy, with hypertension, and co association with proteinuria is shown to be associated with greater adverse maternal and fetal outcome. In pregnant women, found with no proteinuria , but with only mild chronic hypertension then it is similar to non hypertensive pregnant women as shown by Sibai et al 1983 in his study. If a pregnant women is diagnosed to have hypertension together with proteinuria then the association with poor fetal outcome like an increased rate for small for gestational age pregnancies, increased perinatal mortality and maternal morbidity as shown by Chan et al 2005, Brown et el 1996, Ferranzani et al 1990, Chau and Redman 1992, Lin et al 1982 in their study.

Chua et al 1992 in his study, found that delivery was necessarily within 2 weeks of the onset of heavy proteinuria of 5 gm/day in 88.1% of cases and that in a subset of more preterm pregnancies . Pregnancy can be safely prolonged for up to 4 weeks ,with giving intensive monitoring care for both the mother and the fetus.

Sperati and Fine identified that sulfosalicylic acid was added to urine specimen in order to precipitate all protein for the detection of non albumin proteins, the resultant turbidity is graded upon a scale of 0 to 4+ .Even though sulfosalicylic acid testing, is still used ,semi quantitative and quanlitative testing methods have largely replaced it.

Papanna and his associates, as well as Cote and associates suggested that as there is diurnal variation of 24 hour urine protein collection, it would be best to collect spot urine protein creatinine ratio at same time each day, if it is being used to follow up patients.

Cote and et al in their study, iterated that the spot urine protein creatinine rario had a pooled sensitivity of 83.6 % and specificity of 76.3% using a cut off of >.3 for spot protein creatinine ratio to predict the proteinuria greater than 300 mg per day by 24 hour urine collection method of estimation of proteinuria.

PREECLAMPSIA

Preeclampsia is a characteristic disease of pregnancy,that is featured by a specific syndrome, characterised by variable degrees of dysfunction of placenta ,and is associated with a maternal response, that has the features of systemic inflammation. Hypertension and proteinuria, are considered the hallmark of preeclampsia.

The exact nature of the primary event causing preeclampsia is not known. Changes such as

1. Abnormal and inadequate trophoblastic invasion of the maternal spiral arterioles.
2. Increased sensitivity to vasopressin .
3. Elevated peripheral vascular response .
4. Reduced intravascular volume.
5. Thrombophilic factors.
6. Glomerular endotheliosis in renal system.

The aim of management of preeclampsia is focused, towards the earlier diagnosis that is, at the initial stage of the disease and to ameliorate its progression, in an attempt, so that to, wait till the fetal maturity while preventing maternal complication due to preeclampsia.

According to the National High Blood Pressure Education Programe (NHBPEP) and the American College Of Obstetrics And Gynaecology(ACOG).

Hypertension in pregnancy is defined as , blood pressure of systole of more than or equal to 140mmhg or higher and a blood pressure of diastole of 90 mmhg or higher, after 20 weeks of gestation in a women who was with previously normal blood pressure.

ACOG Classification of Hypertension.

1. Preeclampsia, Eclampsia.
2. Chronic Hypertension of any cause.
3. Chronic Hypertension with super imposed preeclampsia.
4. Gestational Hypertension.

Diagnostic criteria for preeclampsia:

1. Systoilc blood pressure ≥ 140 mmhg

   Diastolic blood pressure ≥ 90 mmhg.

2. Proteinuria ≥ 300mg per 24 hour urine collection or proteinuria greater than or equal to 0.3.gm/mmol of spot urine protein creatinine ratio Dipstick reading of 1+ Proteinuria

Or in the absence of proteinuria, new onset of any of the following like

3. Associated , Thrombocytopenia that is– platelet count less than 100,00/microlitre, or
4. Associated Renal insufficiency like – serum crearinine concentration greater than 1.1 mg/dl or a doubling of serum creatinine concentration in the absence of other renal disease, or
5. Associated Impaired Liver function which included

   Elevated blood concentration of liver transaminases to twice the normal concentration, or

6 Associated Pulmonary edema, or

7 Associated Cerebral or visual symptoms.

Risk factors for Preeclampsia:

It includes

Maternal and

Fetal and

Placental causes.

Maternal Factors like

1. Primigravida, or
2. A h/o Previous preeclamptic pregnancy , or
3. Associated increasing maternal age, or
4. Associated Chronic hypertension or a renal diseases or a
5. Associated Maternal chronic inflammatory conditions like Rheumatologic disease, Systemic Lupus Erythematosis or,
6. Associated Obesity or
7. Associated Insulin resistance, or
8. Associated Preexisting Thrombophilias, or
9. Association of Maternal Susceptibility gene, or
10. Associated Family H/O preeclampsia, or
11. Association of Smoking

Placental and Fetal factors like

1. Poor placentation
2. Associated Multiple pregnancy,
3. Associated Hydatidiform mole,
4. Associated Triploidy18,
5. Associated Trisomy 13,
6. Associated Trisomy 16 mosiac,

PREECLAMPSIA

Proteinuria greater than 300mg/day in a 24 hour urine collection or 1+ by qualitative method associated with hypertension that is blood pressure of systole, of more than or equal to 140/90 mmhg and blood pressure of diastole, of more than or equal to 90mmhg , developing after 20 weeks of gestation.

ECLAMPSIA

Patients with preeclampsia developing convulsion is known as eclampsia.

CHRONIC HYPERTENSION

Hypertension developing before 20 weeks of gestation or else presenting before pregnancy. Documentation of hypertension should be at least on two occasion recorded 6 hours apart.

CHRONIC HYPERTENSION WITH SUPERIMPOSED PREECLAMPSIA

Development of proteinuria for the first time during pregnancy in a pregnant women who is a known case of chronic hypertension.

GESTATIONAL HYPERTENSION

In a previously normotensive and nonproteinuric antenatal women, development of hypertension without proteinuria after 20 weks of gestation.

HELLP

It is a severe form of preeclampsia characterised by features of hemolysis, which includes abnormal peripheral smear, bilirubin more than 1.2 mg/dl, thrombocytopenia i.e platelet less than 100000/mm2 and raised liver enzymes like the values of SGOT value of more than 70 U/L, SGPT value of more than 70 U/L, LDH value of more than 600IU/L.

HELLP incidences 0.5 to 0.9 % of all pregnancy as observed by Geary 1997.

Siba et al in 1997 observed that about 20 percent of HELLP cases occurs with severe form of preeclampsia. There are two forms of HELLP. Complete HELLP has all three components, and the partial HELLP contains one or two of the component as observed by Audibert et al in 1996.

CLASSIFICAITON OF PREECLAMPSIA

Mild Preeclampsia:

Blood pressure of systole, of more than or equal to 140mmhg and , blood pressure of diastole of more than or equal to 90mmhg and associated with proteinuria of more than 300mg in 24 hour protein or 1+ urine proteinuria reading in a qualitative method.

Severe Preeclampsia

Systolic blood pressure of more than 160mmhg and a diastolic blood pressure of more than or equal to110mmhg with proteinuria of more than 5 gm in a 24 hour urine collection method or more than 3+in a qualitative method.

Other manifestation of severe preeclampsia like any of the below mentioned systemic complication like,

- Urine output less than 500ml per day, or
- Associated Cerebral, or visual disturbances, or
- Associated Pulmonary edema or cyanosis, or
- Aassociated epigastric or right upper quadrant pain ,
- Associated Impaired liver function, or
- Asssociated Thrombocytopenia, or
- Associated Fetal growth restriction.

PATHOGENESIS

The manifestation of preeclampsia can be grouped as maternal manifestations and the fetal manifestation. The maternal manifestation include the hypertension and preeclampsia. The fetal manifestation includes the intra uterine growth retardation, oligohydramios and decreased oxygenation.

The preeclampsia can be presenting in a different form.

The early onset preeclampsia can be presenting with maternal and fetal complication.

The late onset preeclampsia, nearing term can present with no involvement of the fetus.

The preeclampsia of a nulliparous women may be different from the preeclamptic women who will be having associated, other systemic complication like multifetal gestation, coexisting vascular pathology and gestational diabetes milletus and so on.

The pathogenesis of preeclampsia can be attributed to the below mentioned causes like.

1) Inflammatory responses
2) Activation of the Endothelial cells

3) Abnormal Invasion of cytotrophoblast of spiral arteries
4) Placental pathology in preeclampsia
5) Pro angiogenic and Anti angiogenic protein in preeclampsia antenatal women

The inflammatory Response

In all pregnant women, pregnancy imparts a series of inflammatory response that too during the later half of the pregnancy.

The debris from the syncytio trophoblast are shed into the maternal circulation, initiates the inflammatory response.

If these Placental debris are more in the maternal circulation, then it can produce a danger signal to the maternal immune response. The point of imbalance between the systemic inflammatory response produced by the placental debris and the innate maternal immune response triggers the preeclampsia.

Redman and et al attributed, that the placental ischemic reperfusion injury secondary to the defective placentation predisposes to the onset of preeclampsia in their two stage model of preeclampsia. In their two stage model, the first stage includes the poor placentation.

The second stage, the systemic inflammatory response explains the reason of preeclampsia in the pregnant women with large placenta.

Activation of the Endothelial Cells

The systemic inflammatory response involves the leucocytes, the clotting system and the complement system that triggers the endothelial cell activation.

The principle organ to be involved in the pathophysiology of preeclampsia is the endothelial cells of the vascular systems as shown by the imbalance between the vasoconstrictors Thromboxane A_2 (Tx A2) and the vasodilator prostacyclin PGI_2, abnormalities in the nitric oxide and cyclic guanosine monophosphate pathway, which are the factors that indicate the endothelial cell activation.

The microscopical and the morphological evidence of endothelial cell injury in preeclampsia are evidence by the glomerular endotheliosis, ultra structural changes in the bed of the placenta and uterine vessels.

The activation of the endothelial cells explains the activation of the platelets and it results in the spiral artery thrombosis and the placental infarction, hence there is a reduction in the uteroplacental blood flow.

The increased platelet derived Thromboxane A_2, Serotonin and a decreased prostacyclin I_2, Nitric oxide explains the activation of the inner linning of the spiral arteries, mediated by the surface platelet activations. These activated platelets adhere to one other and release and dense granules like TxA_2, serotonin which contributes for the platelet aggregations and induces the fibrin formation especially at the uteroplacental bed

Impaired Invasion of spiral Arteries by the Cytotrophoblast

The cytotrophoblast replaces the endothelial cells of the spiral arteries and this invasion reaches upto the inner third of the myomoterium, in normal pregnancy.

This invasion of the cytotrphoblast results in a low resistance arteriolar vasculature, it allows for a increasing blood circulation so as to provide the growing fetus.

At all time, the fetal and the maternal bloods are separated by a layer of syncytiotrophoblasts, a layer of cytotrophoblast, mesenchyme, and the walls of the fetal capillaries.

The fetus thus can secrete substance directly into the maternal blood. But the products from the maternal side has to pass the trophoblast and cytoblast before they reach fetal blood.

At the 12 weeks of gestation of true intervillous membrance is formed.

The natural killer cells produces the cytokines that are involved in the angiogenesis and the stability of the vascular system like VEFG, PLGF angiopoietin 2, and thus it imparts a major role in regulating trophoblast invasion and the maternal placental bed vascular changes.

In this way the hundreds of spiral arteries are transformed by the cytotrophoblast into a dilated, low resistance vascular system.

The physiologic changes that happens in the spiral arteries are confined upto only the decidual portions of the arteries in preeclampsia.

In preeclampsia about 30% to 50% of spiral arteries escapes totally from the endovascular invasion.

In addition, there is occlusion of the arteries by the fibrinoid material and invasion of foam cells.

This acute atherosis and association of thrombosis, predisposes to the placental infarction that are common in preeclampsia.

For this above mentioned factor, the abruption is more common in preeclampsia.

The process of abruption in preeclampsia is initiated by the process of thrombotic lesion in the placental vessels, which leads to the necrosis of the placenta, which leads to the separation of placenta and haemorrhage.

Pro Angiogenic proteins and Anti angiogenic proteins in preeclampsia.

The Vascular Endothelial, Growth Factor – a and Placental Growth factor are produced by the cytotrophoblasts of the villous and extravillous membrance, syncytotrophoblast and decidual leucocytes.

The VEGF and PLGF are responsible in inducing vasodilators like prostacyclins I_2 and Nitric oxide in the endothelial cells, to produce vasodilatation during pregnancy and these factors are responsible for the increased glomerular filteration rate during pregnancy.

The fms like tyrosine kinase – 1 and the kinase insert domain containing receptors are the receptors for the VEGF – A and are expressed on trophoblasts and endolhelial cells.

The alternative splicing of the Flt-1 gene creates the soluble fms like tyrosine kinase – 1, which is a principle inhibitor of endogenous angiogenesis inhibitor. Since it has the ability to bind with VEGF and PLGF but it prevents the binding of VEGF, PLGF with the cell receptors. This results in the deficiency of the free VEGF, PLGF, that ultimately leads to the state of endothelial dysfunction.

During the early pregnancy, there is over expression of proangiogenic proteins and towards nearing term there is overexpression of antiangiogenic factors. In preeclampsia, there is upregulation of messenger RNA to s fms- like tyrosine -1, that causes a raised level of s-fms like tyrosine -1 in systemic circulation.

The raised level of s fms-1 and the soluble form of Endogin (sEng) are found to be increased in preeclampsia women.

The soluble form of Endoglin and the sfms- like tyrosine -1 acts together to accelerate the dysfunction of the placenta and causes the

clinical features of preeclampsia, which includes a syndrome of HELLP or a IUGR.

The soluble form of Endoglin / sEng, are found to raised even 6 to 10 weeks before the onset of preeclampsia.

Recently a new human specific splicing variant of VEGF receptor-1 termed as s-fms14 is discovered, which is a potent VEGF inhibitor.

The Systemic Changes in preeclampsia.

Cardiovascular System

In preeclampsia, there is a 30 – 40% fall in plasma volume in a case of severe preeclampsia and 10% fall of plasma volume in case of mild preeclampsia. This results in hypovolemia.

The characteristic feature of severe preeclampsia is vasoconstriction and the leaky capillaries which facilitates the movement of fluid from the intravascular compartment to the extra cellular interested space, resulting in hypovolemia and hemoconcentration.

The increased microvascular permeability to the plasma proteins, a decrease in the osmotic pressure of the plasma colloid and an interstitial protein mass increase are the cause of pathological edema of preeclampsia.

In preeclampsia of severe and early onset, there is a cross over of high cardiac output and low systemic vascular resistance in the earlier stages to the low cardiac output and high systemic vascular resistance the later stages.

Abnormal Hemostasis

The activation of platelet is accelerated in preeclampsia, which compared to normal pregnancy. There is a local grade of compensated intravascular coagulation due to adherence of platelet at the sites of damage of endothelial cells.

In mild preeclampsia a 7% incidence of thrombocytopenia and there is a incidence of 30% to 50% of thrombocytopenia in severe preeclampsia.

Most of 90% of cases of thrombocytopenic preeclampsia women shows a improvement in the platelet count by the 4^{th} postpartum day. While women with severe thrombocytopenic shows a increase of thrombocytes by the 10^{th} postpartum day.

The Liver

Large deposits of fibrin like material obstruct blood flow in the hepatic sinusoids and this causes hepatic capsular distension which is the cause of upper epigastric region pain in the case of preeclampsia.

It is observed that 90% of the HELLP patients have epigastric region tenderness or pain.

The HELLP syndrome patients can present with epigastric pain, shoulder pain, shock massive ascites, pleural effusion and respiratory distress due to the liver rupture or subcapsular hematoma.

If the sub capsular hematoma ruptures within the capsule the patient is hemodynamically stable.

The subcapsular haematoma can be picked up the ultrasonography and the CT scan.

The commonest site of hematoma formation is on the anterior surface or the inferior surface of the right lobe of liver.

The Brain

The central nervous systemic manifestation of the preeclampsia and eclampsia are headache, hyperreflexia, clonus, visual changes, mental changes tinnitus, drowsiness, seizure.

The convulsions occurring in eclampsia are grand mal in character which has got both the tonic and clonic phases. The hallmark of preeclampsia are clonus and hyper reflexia.

The warning sign of an impending eclampsia is also a clonus.

The spasm of the retinal arteriolar system, the ischaemia, and edema are the causation of visual disturbances like blurring of vision, diplopia, scotomas, chromatopsia, hemianopsia.

Retinal detachment can also cause altered vision.

In preeclamptic women, the middle cerebral artery (MCA), posterior cerebral artery (PCA), blood flow and cerebral perfusion pressure are increased which results in hypertensive encephalopalthy and over perfusion of brain.

There is cerebral capillary leak and a resultant vasogenic edema combined with the overperfusion results in local areas of infarction in the preeclamptic women.

The multiple microinfarction and microhaemorrhafes and edema in the occipital gray matter results in the cortical blindness. The posterior reversible encephalopathy syndrome is significant to eclampsia. The features of papilledema signifies cerebral edema in preeclampsia.

IMMUNOLOGY OF PREECLAMPSLA

The trophoblastic invasion into the decidua and into the myometrium, is found to be under the control of the immune mechanisms. The class human leucocyte antigen are not expressed by the

syncyto trophoblasts. The cytotrophoblast express the HLA – G, HLA – E, HLA-C. Earlier it was thought that the HLA–G, played a significant role in the maternal tolerance to the fetus and it was because of HLA- G the fetus was not perceived as “foreign”. Now the present studies shows that the expression of the HLA- G in the cytotrophoblast is essential for the requirement of the vascular adaptation in the bed of the placenta. It was believed that the T cells were the immune cells that were responsible for the adaptations of the immunological responses, in preeclampsia. The absence to be the reason for the immune maladoptation hypothesis.

The decidua contains lymphoid cells that mainly belong to the group of Natural Killer cells, the T lymphocytes and B Lymphocytes are rare. The inhibitory and activatory killer cell immunoglobulin like receptors are expressed in Natural killers cells, that are able to recognize the HLA - Class I molecule. These placental natural killer cells has got influence on both the trophoblast and on the maternal placental bed vascular changes by producing cytokines, that are responsible for angiogenesis and vascular stability.

There are so many Natural Killer, killer cells immunoglobulin like receptor haplotypes in the human being. The expression of killer cell immunoglobulin on the decidual natural killer cells of HLA – C and since

the HCA- C is polymorphic in each pregnancy. There is involvement of differently arranged grouping of the paternally derive fetal HLA – C and maternal KIR.

Hence each and every pregnancy is basically dependent on the unique couple specific Natural Killer cells interactions with the HLA of the paternal origin. Mothers who are lacking the many, or all of the activating killer cell immunoglobulin likes receptor, and when the fetus had HLA – C are at significant risk to develop preeclampsia.

The sperm cells also contributed to the main part in the protection of previous sperm exposure.

There is a release of so many number of cellular and molecular events which resembles the inflammatory response during coitus.

The transforming growth factor – b1 (TGF b1) appears to be a critical seminal factor.

This transforming growth factor – b1 increase the capacity to sample ands process paternal antigens that are in the ejaculate.

The antigen presenting cells, processes the paternal antigens and it will initiate a type 2 immune response.

This type 2 immune response against the antigens of the paternal ejaculate and transforming growth factor – b1, might inhibit the induction of type 1 responses towards the seminallogenic concepts which are responsible for the poor placental and fetal development.

HYPOTHESIS OF PLACENTAL DEBRIS AND ISCHEMIA OF THE PLACENTA

The syntotrophoblats microvillous membranes, the free fetal DNA, and the Cytokeratin are the circulating debris of the placenta. These circulating debris of the placentas are thought to play an important role in the systemic inflammatory response associated with normal pregnancy and preeclampsia pregnant women.

In the future preeclamptic women, it is identified that there is an increased placental debris that are detectable at 16 to 18 weeks.

The presence of elongated syncytial sprouds on along pedicles explains the increased syncyto trophoblabt debris in preeclamptic.

In the formation of syncyto trophoblast debris from the villous cytotrophoblast apoptosis plays a main role.

In preeclampsia, apoptosis is increased, the maternal blood of a preeclampsia women has more particles.

In the preeclamptic pregnancy, during the early onset of preeclampsia, there is increased apoptosis which is explained by the tumor necrotic factor (TNF) interferon gamma (INF-□) fas ligand.

The other placental factors are also found to be increased in their levels in a preeclamptic women. The other placental factors are inflammatory cytokines corticotroptin releasing hormones activin –A.

These maternal inflammatory response syndrome and placental ischaemia reperfusion injury are found to be the reason for the oxidative stresss to preeclampsia.

HYPOTHESEIS OF CONFLICT OF GENETICS

The theory of genetic conflict by Haigs state that the fetal genes will be programmed to increase the transfer of the nutrients to the fetal circulations and the maternal genes will be programmed in such a way to decrease the transfer of nutrients to the fetal circulation in excess of maternal optimum.

The term genomic imprinting means that inside the fetal cells, there exists a difference between the genes of the maternal and the paternal origin.

This hypothesis of conflict predicts the fetal genes will be acting to increase the maternal blood pressure, the maternal factor will be acting to reduce the maternal blood pressure.

The placental factors will be acting in the way that it increase the non placental resistance as the uteroplacental arteries are changed and modified to a condition, so that it remains non reactive the vasoconstrictor. The intrinsic effect of a high maternal systemic blood. psressure are ultimately beneficial to the fetus.

Thus the hypothesis of conflicts of genetics says that the fetal genes will improve the blood flow to the maternal system through the

intervillous space by increasing the maternal blood pressure. So as per the hypothesis of conflict of genetics, the blood pressure of the women is balanced by the fetal factors the increase the blood pressure and maternal factor the decrease the blood pressure.

The correlation between the angiogenic growth factor and soluble fms like tyrosine – I particles provide the example of molecular pathway.

As the soluble fms like tyrosine – 1 increase the severity of disease increase which further supports, that VEGF particularly soluble fms like tyrosine – I particle balance, represents a main final common pathophysiologic pathway for preeclampsia.

PREDICTION OF PRECLAMPSIA

Predictive test are the test that are used for predicting women, who will develop preeclampsia and it should be simple, rapid, noninvasive ,inexpensive, it should be easy to perform and should not expose the patient for a discomfort or risk.

- Uterine artery Doppler velocimetry.
- Soluble fms like tyrosine kinase -1.
- Soluble endoglin.
- Placental growth factor.
- Vascular endothelial growth factor.
- Placental protein 13.

As of 2012 no single test is reliable in predicting preeclamsia .The current evidences suggest that a combination of all these biomarkers, along with uterine artery Doppler studies can provide the best predictive accuracy for the identification of early onset of preeclampsia.

PROTEINURIA

Proteinuria is one of the diagnostic criteria of preeclampsia. It has been suggested that proteinuria is a caused by two mechanisms.

1) The first cause is thought to be due to the abnormal trans glomerular passage of proteins because of increased permeability of the glomerular capillary wall and also due to the impaired reabsorbtion by the epithelial cells of the proximal tubules in kidney.

2) **The kidney in Pregnancy**

   There are both anatomical and physiological changes in the urinary tract during pregnancy. The kidney during pregnancy enlarges ,the pelvi calycael system, ureter dilates due to both humoral and also due to the obstructive causes .The estimated glomerular filteration rate and the estimated renal plasma flow is found to increase by

50%. Creatinine clearance is found to increase by 4th week and peaks at 9 to11 weeks of gestation and then is sustained until the 36 weeks of gestation. In last four weeks of pregnancy ,creatinine clearance is e reduced by 15-20%.

Significant proteinuria as defined by ISSHP is proteinuria of >300mg/dl in a 24 hour urine collection method or spot urine protein creatinine ratio as more than 0.3.

RECOMMENDED METHOD OF TESTING URINE FOR PROTEIN

Dipstick Method.

All antenatal women are routienly screened for proteinuria during their first and regular antenatal visit by dipstick method or sulphosalicylic method. If the test are found to be positive then further laboratory investigation is necessary.

Dipstick Method

The most commonly used test for quantifying urine proteinuria is the urine dipstick testing. The dipstick usually carries a reagent strip that is impregnated with a Ph indicater. Usually the chemical dye, the tetrabromophenol and a buffer to maintain a Ph of 3.0 . Proteins, usually

the albumin binds to the Ph indicater dye that will produce a change of the colour. This change is independent of urine Ph.

Urine protein:

Trace – 0.1 gm/l

1+ - 0.2 gm/l

2+ - 1 gm/l

3+ - 3 gm/l

4+ - 10gm/l

In 1994 Meyer et al has reported that about 66% of false negative rate for a trace dipstick or absent test result. They also reported 26% false positive rate for dipstick test >1+ for predicting proteinuria of >300mg per day.

QUANTITATIVE ASSESMENT OF PROTEINURIA

Further evaluation of proteinuria is required if persistent proteinuria in dipstick method.

Assessment of quantitative excretion of protein is performed usually on timed collection , a 24 hour urine collection method proteinuria. Gold standard in diagnosis of proteinuria in a preeclampsia is the estimation of proteinuria in 24 hour urine collection for many years. But it is cumbersome for both the patient and the staff and is subject to error due to inaccurate timing and it requires patients compliance. Waiting for the result of protein estimation in a 24 hour urine collection

can delay the diagnosis of preeclampsia unnecessarily potentially put the mother and the fetus at risk .

DIPSTICK TO 24 HOUR URINE PROTEINURIA

The main factors for the usage of the dipstick test is that the test can be done easily and the low cost of the test. Proteinuria of 1+ by dipstick analysis method is believed widely to correspond to 300mg/24 hour urine protein collection method by. Dipstick testing is associated with false positive results which is mainly due to concentrated urine i;e specific gravity of more than 1.030, alkaline urine contaminated with vaginal discharge. Brown et al in his study has reported 8-18% of false positive and a 67% of false positive cases with 1+.

Waugh et al in his study on 197 preeclampsia women observed and found a 65% of the 197 pregnant preeclamptic women had a false negative urine proteinuria in women with >1+ proteinuria in dipstick method, but with significant proteinuria.The statistical datas of the above study showed that there is a poor correlation between the dipstick and 24 hour urine method . Thus reviewing the literature shows that the accuracy of urine analysis is inadequate in predicting proteinuria significantly.

SPOT URINE PROTEIN CREATININE RATIO WITH 24 HOUR URINE COLLECTION METHOD

In a study done and analysed by Shahbazian et al among 80 pregnant women with preeclampsia, he observed that there was a strong correlation, between the spot urine protein creatinine ratio and 24 hour urine collection. The cut off ratio of spot protein creatinine to 24 hour ratio is 0.3 for 300mg/24 hour with a positive predictive value of 94.45,negative predictive vlue of 96.8% sensitivity of 91.2%, specificity of 87.8%The value of less than 0.29 yeiled a sensitivity of exclusion of preeclampsia by 100%.

Eigbeoh and et al conducted another study and observed that there was a strong correlation between the spot protein creatinine ratio and 24 hour protein ratio(the coefficient correlation r=0.823,p value <0.001) and they found that in all case 24 hour urine collection was more cumbersome and can be replaced by the easy and convenient spot urine protein creatinine ratio. Cut off for posiiive spot protein creartinine ratio have been standardised by the International Society for the Study of Hypertension (ISSHP) as .3 in 2001 and a systematic review and a meta analysis of articles from 1997 through 2008 confirmed this cut off for adequate sensitivity and specificity and described the use of the spot PCR as promising.

Papanna Ret al in 2008 conducted a study to estimate the accuracy of the spot urine protein creatinine ratio in predicting 300 mg of significant protein excretion in 24 hours urine collection in pregnant patients with preeclampsia, through a systematic review that included 21 studies. From the 21 studies they concluded that random spot urine protein creatinine ratio determination are helpful primarily, when they are below 130 -150 mg/dl in that 300mg or more proteinuria is unlikely to below this threshold.

Bhavana et al in 2009, conducted a study in the hospitalised pregnant women with preeclampsia and the aim of the study was to compare the spot urine protein creatinine ratio in single voided urine sample with that of the 24 hour urine protein for estimation of proteinuria . In their study they came to a conclusion and observed that there was a significant correlation between 24 hour protein, and found that spot urine protein creatinine ratio appeared to be an excellent alternative to 24 hour urine protein.

Leonas Miranda et al in 2007 in a cross sectional study of 927 hospitalised pregnant women with preeclampsia and in a 2nd cohort of 1161 pregnant women in whom hypertensive disorder of pregnancy was ruled out for comparison found that the spot urine protein creatinine ratio

and 24 hour urine excretion were significantly correlated r = 0.98, p <0.001. The spot urine protein creatinine ratio as an indicator of protein excretion >300mg/24 hour was >/0..3 . The sensitivity and specificity were 98.2% and 98.8%. The positive and negative predictive likely hood ratio were 79.2and 0.92 respectively.

They concluded that the spot urine creatinine ratio is a reliable indicator of significant proteinuria >300mg/24 hour. The spot PCR may be used as an alternatively to the 24 hour urine collection method.

Celesta Durnwald , Brian Mercer (2003) in their prospective case study done to determine the values of spot urine PCR, in prediction of 24 hour urine proteinuria among the patients with preeclampsia . In their study a total of 220 women were evaluated on significant and severe proteinuria on 24 hour urine protein evaluation were indentified in 76.4% and 8.2% of cases respectively . In their study the regression analysis of spot urine PCR and 24 hour urine proteinuria showed a coefficient correlation of r= 0.4.

Detecting significant proteinuria by using spot PCR as a substitute to 24 hour excretion remains unclear. Many investigators have proposed the use of spot urine protein creatinine ratio, but still there are conflicting results.

In this study in patients with preclampsia admitted for evaluation ,the correlation between the spot PCR to 24 hour urine proteinuria was analysed.

AIM OF THE STUDY

The correlation between the 24 hour urinary collection to

1) To compare and correlate between the spot urine protein creatinine ratio in a single voided urine sample with the 24 hour urine protein ratio for estimation of proteinuria in patients with preeclampsia.

2) To know if the the spot protein creatinine ratio can be used to quantify the proteinuria accurately and rapidly and at the time can overcome limitation of the routinely performed test.

OBJECTIVES OF THE STUDY

To compare spot urine protein creatinine ratio in a single voided sample with 24 hour urine protein ratio for estimation of proteinuria in preeclampsia.

MATERIALS AND METHODS

1) STUDY PERIOD : 1 YEAR
2) SAMPLE SIZE : 150 CASES
3) STUDY DESIGN : PROSPECTIVE STUDY
4) SOURCE OF DATA
5) This study was done at the department of Obstetrics and Gynaecology at the Institute Of Social Obstetrics, Government Kasturba Gandhi Hospital attached to the Madras Medical College after getting Ethical clearance from the Hospital Ethical Clearance.
6) This study was done in 150 antenatal patients who had been admitted for evaluation of preeclampsia prospectively after explaining the nature of study and getting informed written consent.
7) Selection criteria
8) Antenatal women with preeclampsia of blood pressure of equal or more than 140/90 mmhg recorded on two occasions at least 6 hours apart , after 20 weeks of gestational age with previously normal blood pressure with proteinuria defined as urinary excretion of >300mg/day protein or higher in a 24 hour urine specimen .
9) Patients were categorised as severe preeclampsia as mentioned earlier in the classification of the preeclampsia

INCLUSION CRITERIA

- Patients with BP ≥ 140/90mmhg.
- Patients with > 1+ proteinuria.
- Singleton pregnancy.
- Gestational age >20 weeks.
- No H/O renal disease.
- No H/O hypertension

EXCLUSION CRITERIA

- Case with gestational age <20 weeks.
- Known hypertensives.
- Known renal diseases.
- Known epileptics.
- Multiple pregnancy.
- Gestational diabetes.
- UTI.

PROCEDURE

Study was undertaken in one hundred and fifty patients who satisfied the above criteria. After explaining the procedure and purpose of study informed consent was obtained from all the patients. All the antenatal women with the elevated blood pressure and proteinuria were admitted after excluding the above mentioned excluding criteria and the admission blood pressure was recorded for all the patients in the right upper limb in sitting posture at level of the heart. Diastolic BP was determined as the disappearance of Korotkoff sound V. A detailed obstetric history was elicited. General examination and systemic examination was done for all the patients. Complete obstetric examination was done.

Admission blood pressure was checked again after 6 hours. One hundred and fifty patients with elevated blood pressure and elevated porteinuria were included in this study.

Urine for urine routine analysis and urine culture sensitivity was sent to the lab to rule out infection. A random sample of urine proteinuria was assessed by the dipstick method grading of proteinuria by dipstick is as follow.

Trace - 0.1 gm/l

1+ - 0.3 gm/l

2+ - 1 gm/l

3+ - 3 gm/l

4+ - 10gm/l

Patients urine which shows proteinuria of 1+ by dipstick were analyzed for the spot urine protein creatinine ratio and 24 hour urine protein ratio.

A spot mid stream sample of urine was collected from all the patient before the collection of 24 hour urine protein collection estimation was done.

Modified Jaffes method was used to estimate the urine creatinine and calorimetry method was used to estimate the urine protein.

The spot urine protein creatinine ratio was obtained by dividing the urine protein concentration in mg/dl by the urine creatinine concentration in mg/dl.

24 hour urine samples were collected after collecting the specimen for the spot test and the 24 hour urine estimation was done.

Normal values of proteinuria in preeclampsia

In 24 hour urine proteinuria

Significant proteinuria	>300mg/day
Proteinuria of severe range	>5000mg/day

Spot urine protein creatinine ratio:

Insignificant	<0.3
Significant	>0.3

Complete blood count ,which included haemoglobin in mg/dl ,platelet count , Renal function test that included blood urea, serum creatinine, and Liver function test which included serum bilirubin, SGOT,SGPT Serum proteins like serum albumin, serum globulin.

Examination of the fundus of the eye was carried out to all patients.

For cases which had suspicion of IUGR, ultrasonogram and Doppler study was done wherever indicated to confirm the same.

The collected data were analyzed by using appropriate statistical methods.

The relation between the spot urine protein creatinine ratio and the 24 hour urine proteinuria was assessed by the Pearson correlation test.

RESULT AND ANALYSIS

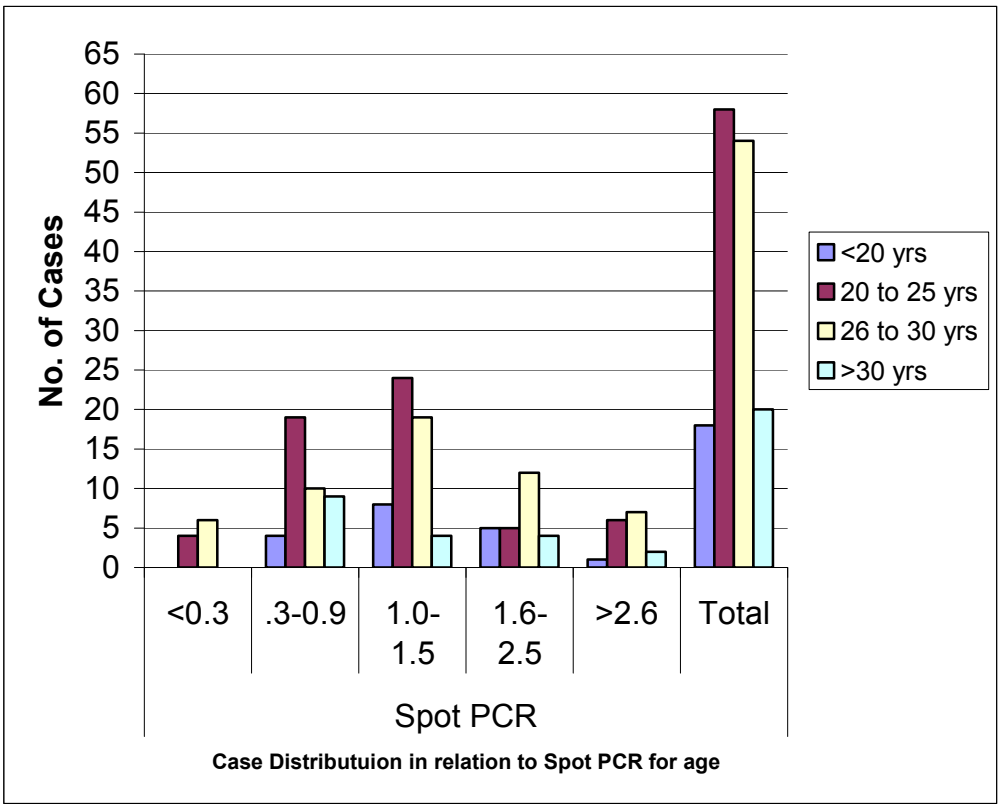
TABLE I AGE DISTRIBUTION COMPARISON WITH SPOT PCR

S. No	Age	Spot PCR					Percentage
		<0.3	.3-0.9	1.0-1.5	1.6-2.5	>2.6	
1	<20 yrs	0	4	8	5	1	12
2	20 to 25 yrs	4	19	24	5	6	38.7
3	26 to 30 yrs	6	10	19	12	7	36.1
4	>30 yrs	1	9	4	4	2	13.3
	Total	11	42	55	26	16	100

Table I shows the age distribution of 150 cases studied and it depicts that majority of case fall in the age group of 20. About 38.7% of cases were in the age group of 20 to 25 yrs.In the age group of 26 to 30 yrs there were36% % of cases.There were 12 % of cases in the age group less than 20 yrs.There were 13.3% of cases in the age group of more than 30yrs

BAR CHART OF THE AGE DISTRIBUTION

IN NUMBERS



This chart shows the distribution of age frequency in relation to spot PCR .

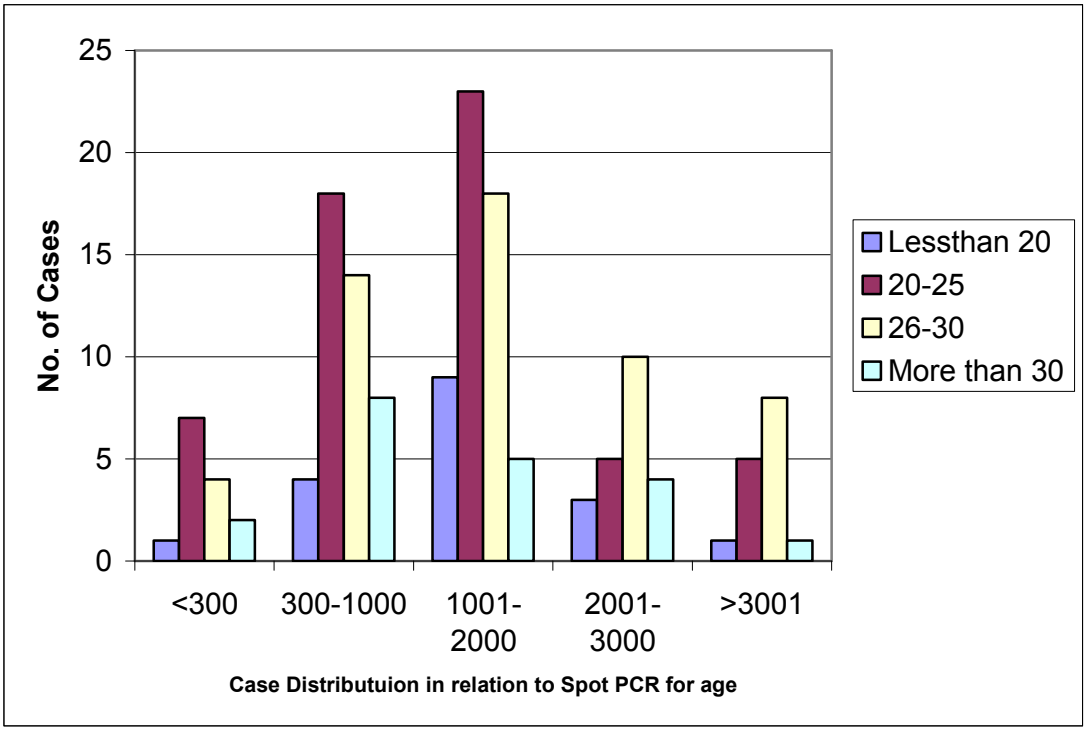
In this study there were 18 cases less than 20yrs of age, there were 58 cases in age group of 20-25 yrs,there were 54 cases in the age group of 26 to 30 yrs, and 20 cases in the age group of more than 30 yrs.

TABLE II COMPARISON OF AGE FREQUENCY WITH 24 HOUR URINE PROTEIN RATIO

S.No.	Age group In yrs	24 hour urine protein ratio in mg //day					Percentage
		<300	300-1000	1001-2000	2001-3000	>3001	
1	<20	1	4	9	3	1	12
2	20-25	7	18	23	5	5	38.6
3	26-30	4	14	18	10	8	36
4	>30	2	8	5	4	1	13.3
	TOTAL	**14**	**44**	**55**	**22**	**15**	**100**

This table shows the age distribution of 150 cases studied in relation to 24. there were 12% of case < 20 yrs. Of age, 38.6% of cases between 20 – 25 yrs, 36% of cases between 26 – 30 yrs, and 13.3% of cases more than 30 yrs.

BAR CHART COMPARISON OF AGE FREQUENCY WITH 24 HOUR URINE PROTEIN RATIO



This Bar Chart Shown the distribution of age frequency in relation to 24 HUP. In this study there were 18 cases < 20 years, 58 cases between 20 – 25 yrs, 54 cases between 26 – 30 yrs and 20 cases > 30 yrs.

TABLE III CASE DISTRIBUTION IN RELATION TO SES

S. No.	SES	Total No. of Cases
1	3	1
2	4	29
3	5	120

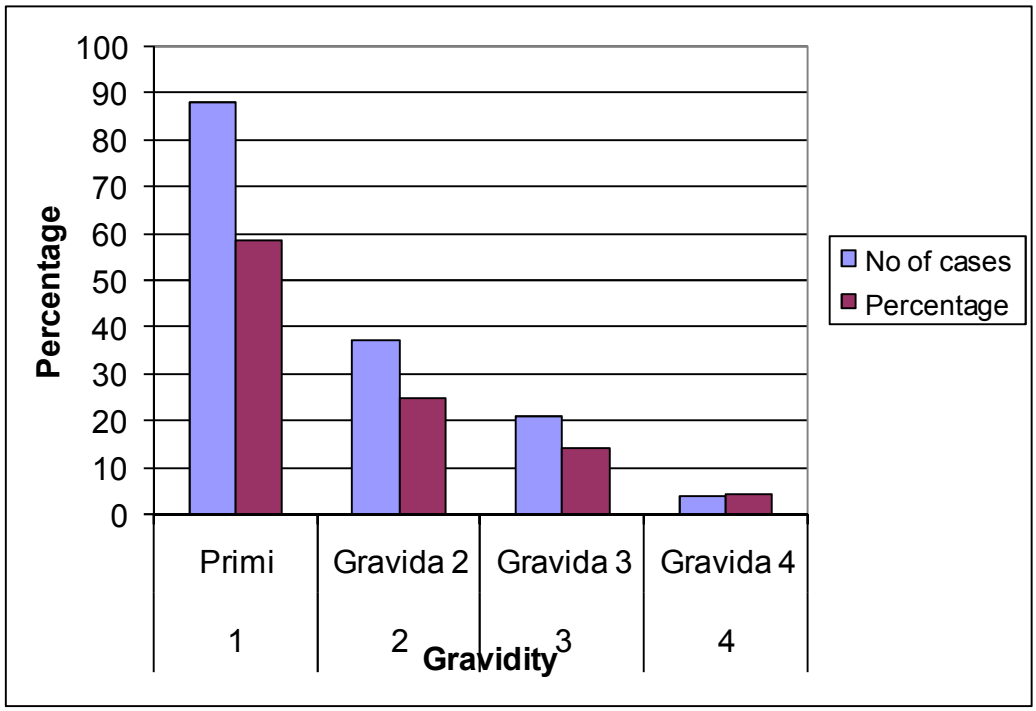
Table III Shows the socioeconomic status distribution frequency. In this study there 120 cases that belonged to socioeconomic status of class V ,and there were 29 cases belonging to classIV and 1 case belonging to class III .This data shows that our hospital cares for the very low socioeconomic status.

TABLE IV CASE DISTRIBUTION IN RELATION TO GRAVIDITY

S.No.	Gravidity	No of cases	Percentage
1	Primi	88	58.6
2	Gravida 2	37	24.6
3	Gravida 3	21	14
4	Gravida 3	4	4.3

Table IV shows gravidity distribution of 150 cases .In this study 58.6% of cases were Primigravida which signifies, the incidence is more in Primigeavida. 24.6% of case were 2nd gravid.14% of cases were 3rd gravidas .1.3% of case were 4th gravidas.

BAR DIAGRAM OF CASE DISTRIBUTION IN RELATION TO GRAVIDITY



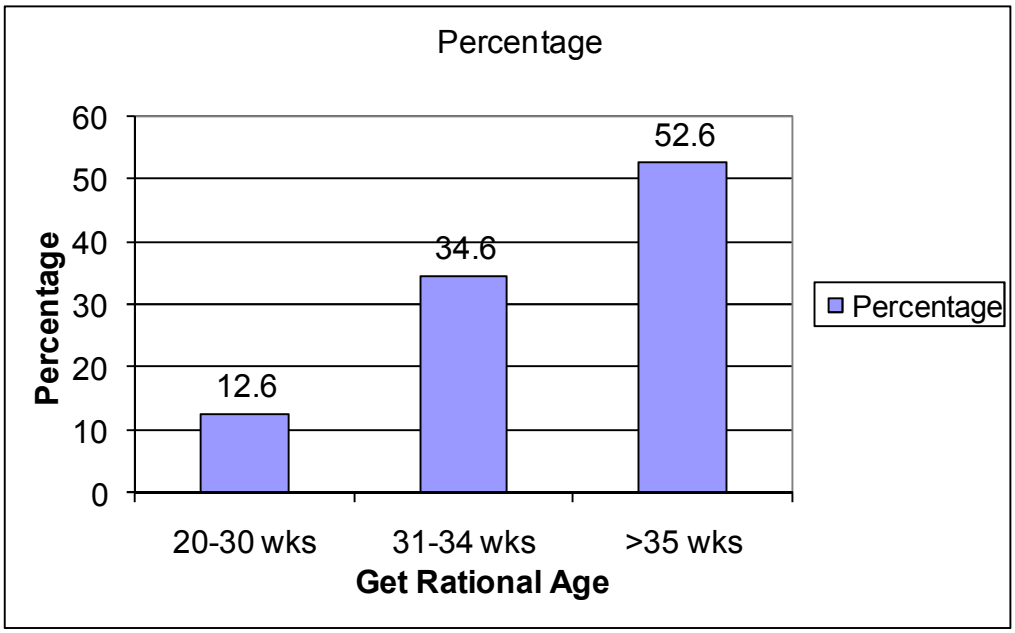
This bar chart shows the gravidity distribution of 150 cases studied.In this study there were 88 cases of primigravida, 32 cases of 2nd gravid, 21 cases of 3rd gravid, and 4 case of 4th gravia. This shows that majority of case were primigravida which tells us the more incidence of preeclampsia in primigravida.

TABLE V CASE DISTRIBUTION IN RELATION TO GESTATIONAL AGE

S.No	Getrational Age	No of cases	Percentage
1	20-30 wks	19	12.6
2	31-34 wks	52	34.6
3	>35 wks	79	52.6

This Table V displays the distribution of gestational age of the 150 case studied. There were 12.6% of cases in the gestational age group of 20 to30 wks of gestation. Between the age group of 31 to 34 weeks there were 34.6% of cases In this case study. There were 52.6 % of cases in gestational age group of more than 35 wks.

BAR DIAGRAM OF GESTATIONAL AGE



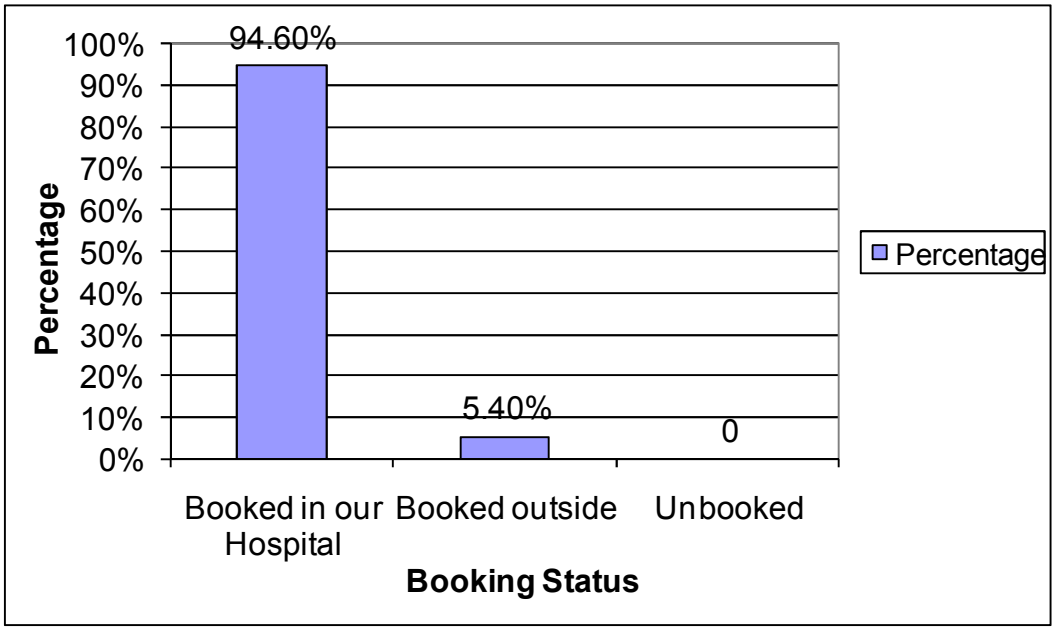
This bar chart shows the distribution of gestational age group frequency in the 150 case studied. There were 19 cases in the gestational age group of 20 to 30 wks., which accounts for 12% there were 52 cases in the gestational age group of 31 to 34 wks. frequency, which accounts for34.65 and there were 79 cases in the gestational age group of more than 34 wks which accounts for 52.6% of cases

TABLE VI DISTRIBUTION OF BOOKNG STATUS

S.No.	Booking status	No of Cases	Percentage
1	Booked in our Hospital	142	94.6%
2	Booked outside	8	5.4%
3	Unbooked	0	0

The above table shows the number of cases booked in ISO KGH and booked outside ISO KGH .94.6% of cases are booked in our hospital. 8% of cases were booked outside our hospital.

BAR DIAGRAM OF BOOKING STATUS



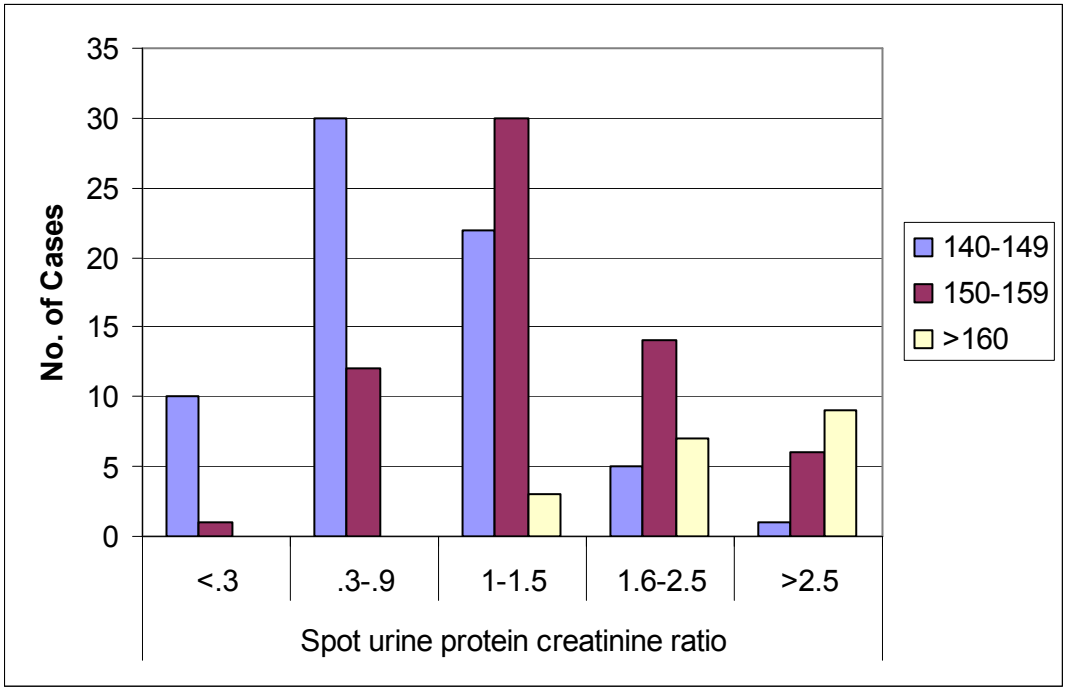
This bar chart shows the booking status distribution of the 150 cases studied. In this study there were 142 cases booked in our hospital and 8 cases were booked outside our hospital .There were no unbooked case. This indicates that preeclampsia was diagnosed in the booked case which insist that the booking was very essential to pick up the cases in the early onset of the disease and avoid the development of the maternal and fetal complication.

TABLE VII CASE DISTRIBUTION IN RELATION TO SYSTOLIC BLOOD PRESSURE AND WITH SPOT URINE PCR

S.No.	Systolic Blood pressure In mmhg	Spot urine Protein Creatinine Ratio					Total
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1.	140-149	10	30	22	5	1	68
2.	150-159	1	12	30	14	6	63
3.	>160	0	0	3	7	9	19
	TOTAL	**11**	**42**	**55**	**26**	**16**	**150**

This Table shows the distribution of systolic blood pressure in relation with the spot PCR. In this study 45.3% of cases presented with systolic blood pressure range of 140 to149 mmhg. 42% of cases presented with systolic blood pressure range of 150 to 159 mmhg. 12.6 % of cases were having systolic blood pressure of>160 mmhg. This shows that majority of the cases were in the mild degree of preeclampsia.

BAR DIAGRAM OF CASE DISTRIBUTION IN RELATION TO SYSTOLIC BLOOD PRESSURE AND WITH SPOT PCR



This bar chart shows the distribution of No. of cases in relation to systolic blood pressure and with the spot PCR. In this study there were 68 cases with systolic blood pressure range of 140 to149 mmhg,63 cases with systolic blood pressure range of 150 to 159 mmhg and 19 cases were in the systolic range of >160 mmhg. In this study there were 11 cases with spot PCR <.3, 42 cases in the range of.3 to .9,55 cases in the range of 1 to 1.5, 26 cases in the range of 1.6 to 2.6,and 16 cases in the range of >2.5.

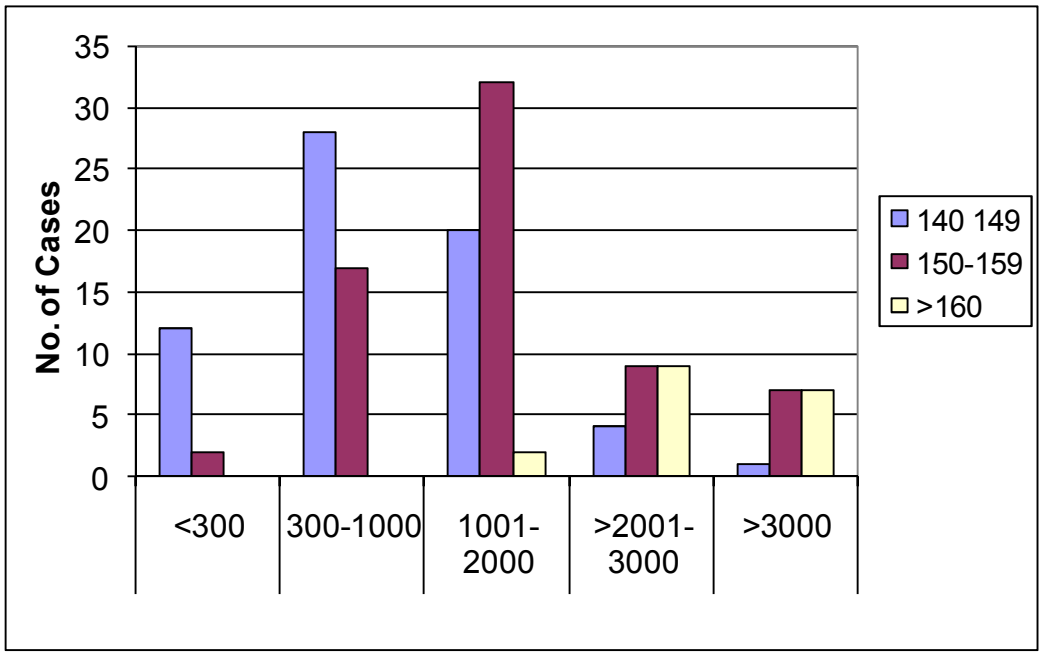
This bar diagram shows the distribution of systolic blood pressure of the 150 cases studied. Most of the cases were in the mild preeclampsia variant and only 19 cases were in the severe preeclampsia variant.

TABLE VIII CASE DISTRIBUTION OF SYSTOLIC BLOOD PRESSURE IN RELATION WITH THE 24 HOUR URINE PROTEIN RATIO

S. No	Systolic Blood Pressure in mmhg	24HOUR URINE PROTEINURIA IN mg/day					Percentage
		<300	300-1000	1001-2000	>2001-3000	>3000	
1	140 149	12	28	20	4	1	43.3
2	150-159	2	17	32	9	7	44.6
3	>160	0	0	2	9	7	12
	TOTAL	**14**	**44**	**56**	**22**	**15**	**100**

In this study 43.3% of cases were having the systolic blood pressure in the range of 140-149mmhg. 44.6% of cases were having the systolic blood pressure in the range of 150-159mmg, 12% of cases were having systolic blood pressure of >160 mmhg.

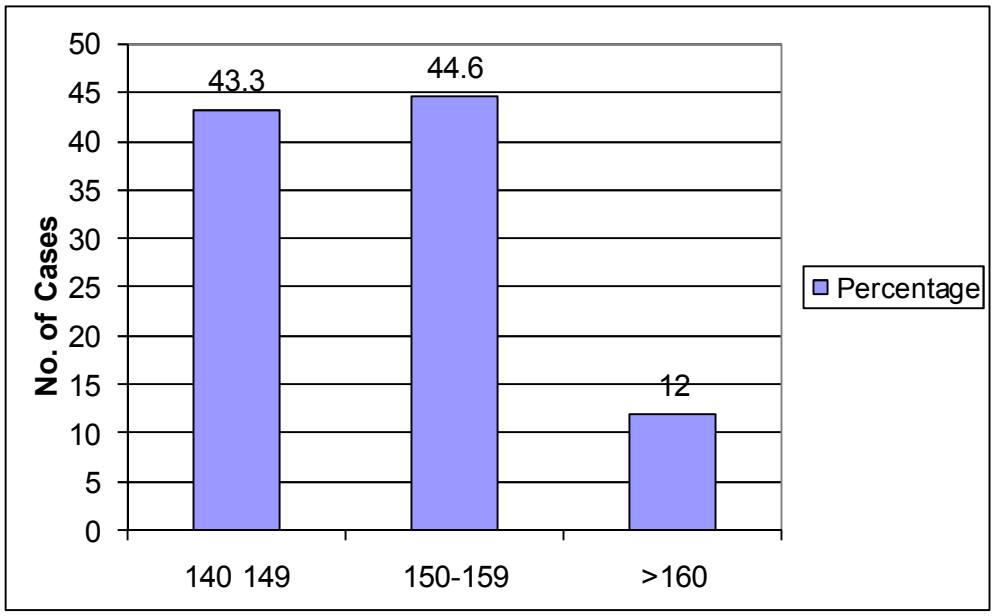
BAR DIAGRAM OF CASE DISTRIBUTION OF SYSTOLIC BLOOD PRESSURE WITH 24 HOUR URINE PROTEIN



This bar chart shows the case distribution of systolic blood pressure and with 24 hour urine protein ratio. In this study there were 14 cases with 24 HUP <300mg/day, 44 cases were in the range of 300 to 1000mg/day, there were 56 cases in the range of 1001-2000mg/day, and 22 cases in the range of >3000mg/day. This shows that most of the cases were only milder form of 24 hour urine proteinuria.

Systolic Blood Pressure in relation to in mmhg

24 Hrs. Urine Proteinuria in mg/day



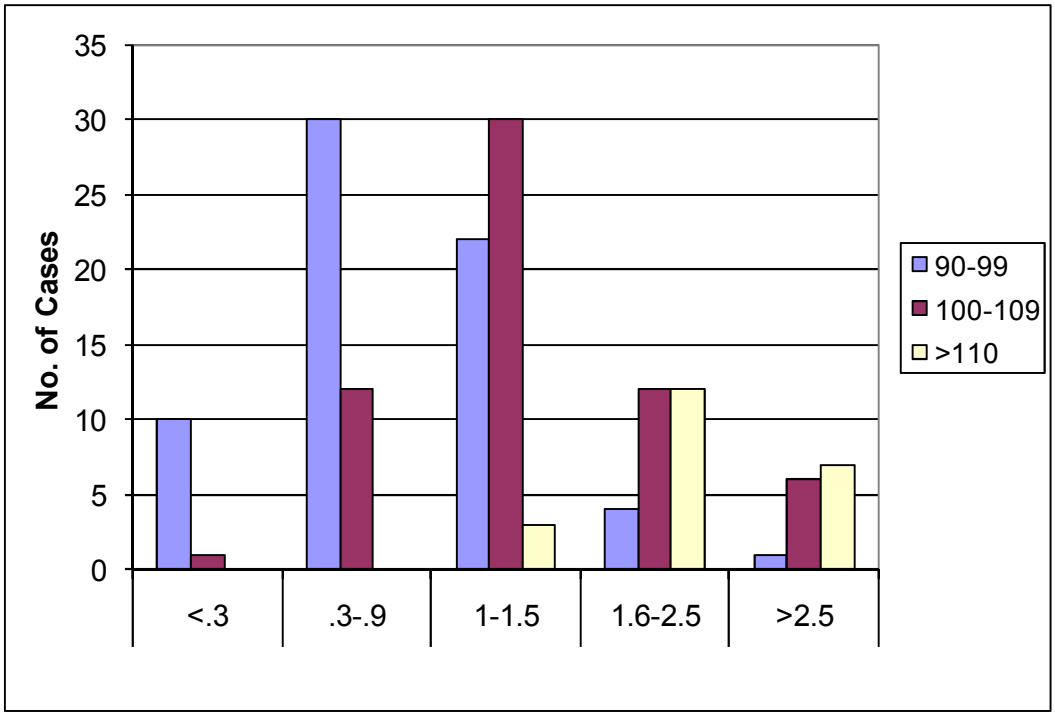
This bar chart shows the number case distribution with systolic blood pressure range frequency in relation to 24 Hrs urine protein ratios. In this study there were 65 cases in the systolic blood pressure range of 14 to 149 mmhg, 67 cases in the range of systolic blood pressure 150 to 159 mmhg, and there were 18 cases in the systolic blood pressure range if >160mmhg.

TABLE IX CASE DISTRIBUTION IN RELATION TO DIASTOLIC BLOODPRESSURE WITH SPOT PCR

S.No	Diastolic Blood pressure in mmhg	Spot PCR in gm/mmol					Percentage
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1	90-99	10	30	22	4	1	44.6
2	100-109	1	12	30	12	6	40.6
3	>110	0	0	3	12	7	14.6
	TOTAL	**11**	**42**	**55**	**28**	**14**	**100**

In this study there were 44.6% of cases with diastolic blood 90 – 99 mmhg, 40.6% of cases with diastolic blood 100 – 109, 14.6% of cases with diastolic blood > 110 mmhg which shows that most of the cases were with in mild preclampsia variant.

BAR DIAGRAM OF DIASTOLIC BLOOOD PRSSURE DISTRIBUTION WITH SPOT PCR



This Table shows the distribution of cases with the diastolic blood pressure range and spot PCR. There were 67 cases with diastolic blood pressure range of 90 to 99 mmgh,61 cases with diastolic blood pressure range of 100 to109 mmhg,and 22 cases in the range of >110mmhg.So most of the case studied were only mild preeclampsia.

There were 11 cases with Spot PCR with .3, there were 42 cases with Spot PCR, .3 to 9, there were 55 cases with Spot PCR 1 – 15, there were 28 cases with 1.6 – 2.5, there were 14 cases with spot PCR > 20.

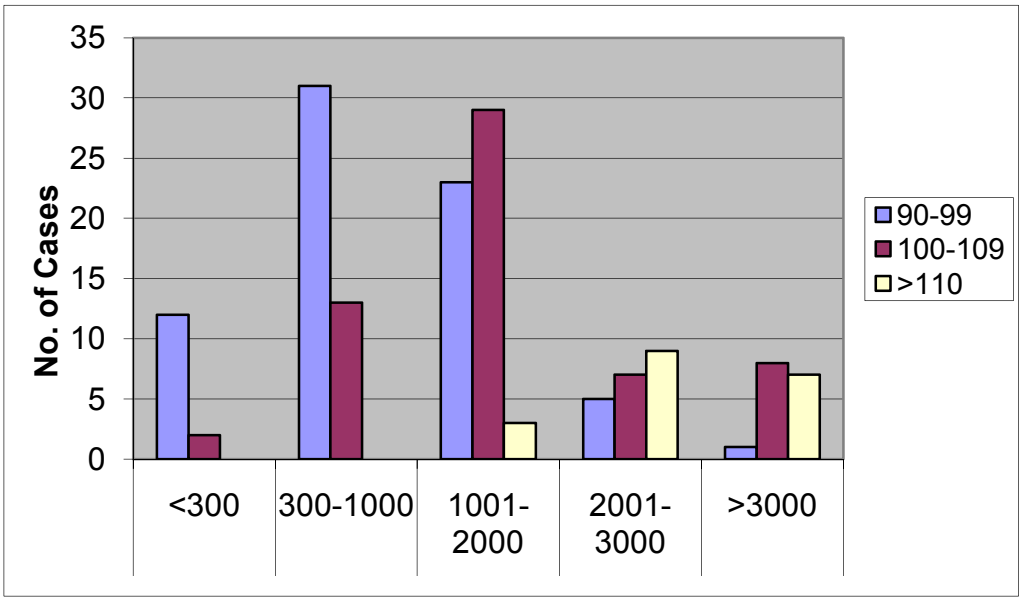
This Bar Diagram shows the number of case distribution with the diastolic blood pressure range. There were 67 cases with the diastolic blood pressure range of 90 to 99mmhg ,there were 61 cases with the diastolic blood pressure range of 100 to109mmhg, and there were 22 case in the diastolic blood pressure range of >110mmhg. Most of the cases were in the mild preeclampsia category in this case study.

TABLE X CASE DISTRIBUTION IN RELATION TO DIASTOLIC BLOOD PREESURE WITH THE 24 HOUR URINE PROTEIN RATIO

S. No	Diastolic blood preesure in mmhg	24 hour urine protein ratio					Percentage
		<300	300-1000	1001-2000	2001-3000	>3000	
1	90-99	12	31	23	5	1	48
2	100-109	2	13	29	7	8	39.3
3	>110	0	0	3	9	7	12.7
	TOTAL	**14**	**44**	**55**	**22**	**15**	**100**

In this case study 48% of cases were having diastolic blood pressure in the range of 90-99mmhg. 39.3% of cases were having diastolic blood pressure of 100-109mmhg. 12.6% of cases were having diastolic blood pressure in the range of >160mmhg

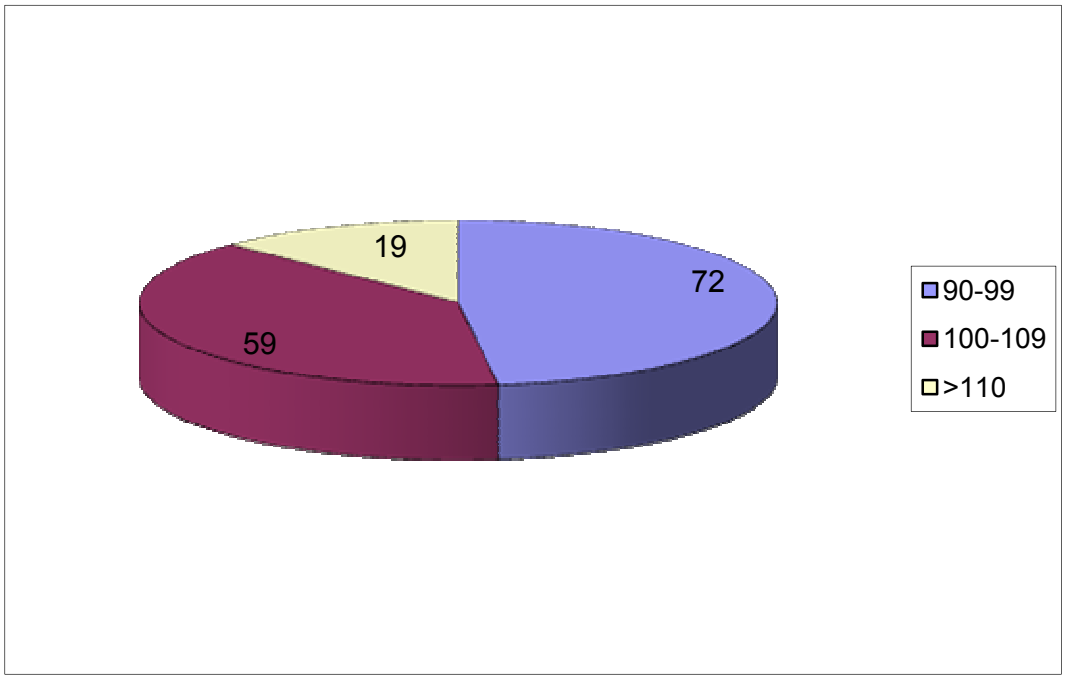
BAR DIAGRAM OF CASE DISTRIBUTION IN RELATION TO DIASTOLIC BLOOD PRESSURE ITH THE 24 HOUR URINE PROTEIN RATIO



In this study this table shows the distribution of diastolic blood pressure with 24 HUP. There were 14 cases with 24 HUP <300mg/day, there were 44 cases with 24 HUP range 300 to 1000mg/day, there were 55 cases with 24 HUP range of 1001 to 2000mg/day, there were15 cases with 24 HUP range of >3000mg/day.

This bar chart shows the No of case distribution frequency with the diastolic blood pressure with the 24 HUP. In this cases study there were 72 case with diastolic blood pressure range of 90 to99mmgh,59 cases with diastolic blood pressure range of 100 to 109mmhg,and there were 19 cases with diastolic blood pressure range of >160 mmhg..

PIE CHART FOR CASE DISTRIBUTION OF DIASTOLIC BLOOD PRESSURE



This pie chart represents the number of case distribution with the diastolic blood pressure range frequency.

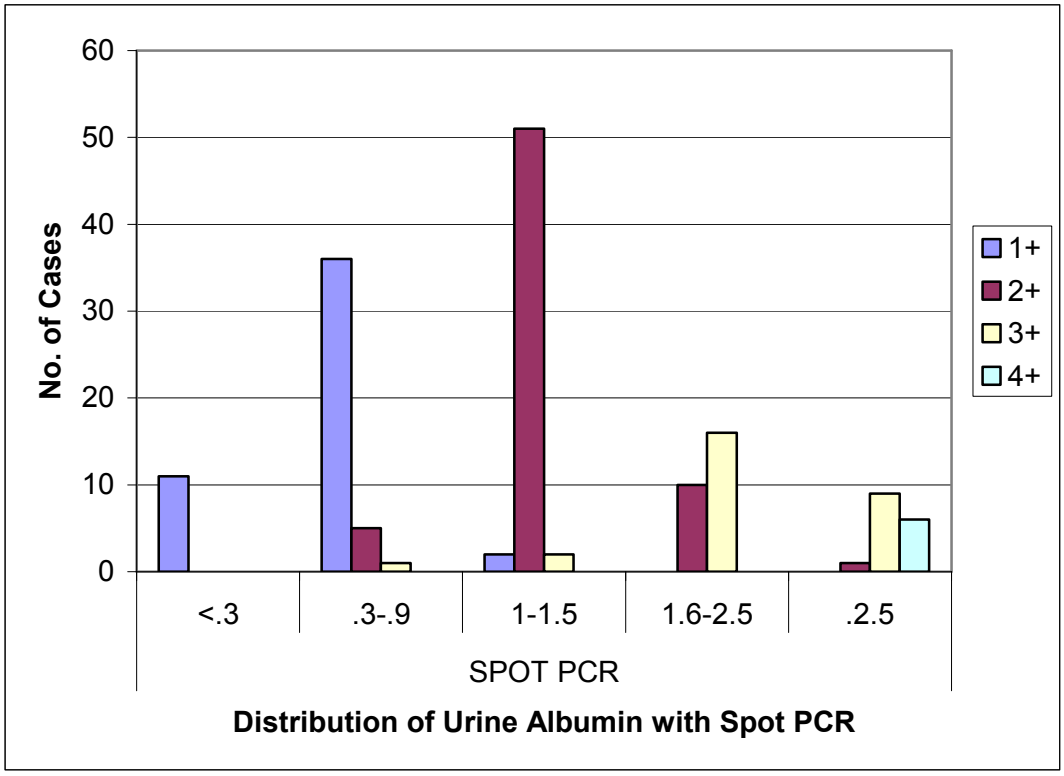
TABLE XI DISTRIBUTION OF URINE ALBUMIN WITH SPOT PCR

S.No	URINE ALBUMIN	SPOT PCR					TOTAL
		<.3	.3-.9	1-1.5	1.6-2.5	.2.5	
1	1+	11	36	2	0	0	49
2	2+	0	5	51	10	1	67
3	3+	0	1	2	16	9	28
4	4+	0	0	0	0	6	6
	TOTAL	11	42	55	26	16	150

In one hundred and fifty cases studied 32.6% of cases were having urine albumin of 1+ 44.6% of cases were having urine albumin of 2+. 18.6 of cases were having urine albumin of 3+. 4% of cases were in the range of 4+ urine albuminuria.

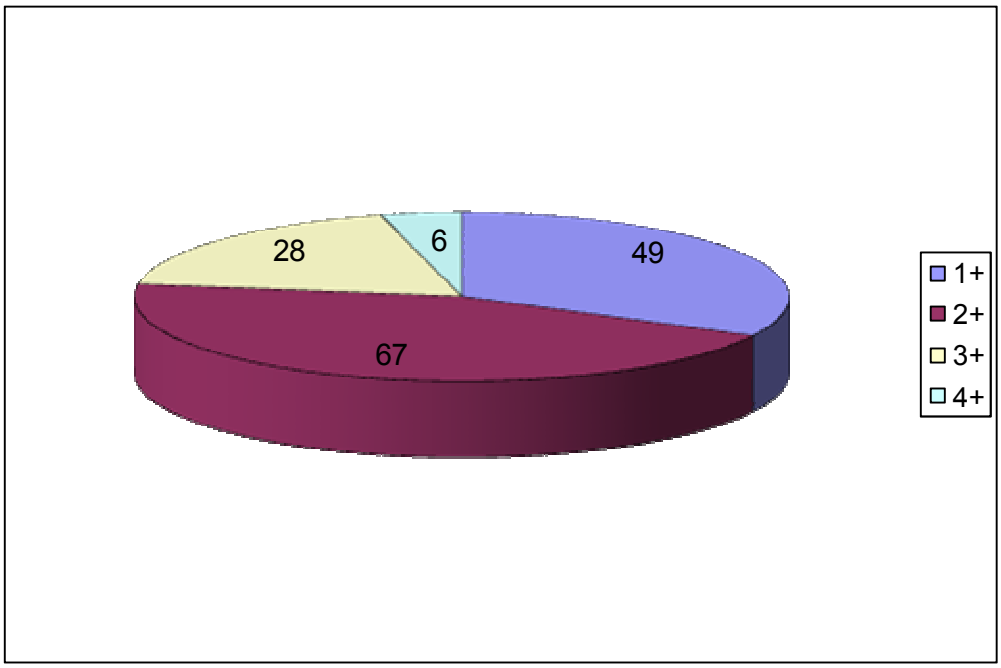
This table shows the distribution of urine albumin with the spot PCR. In this case study there were 11 cases with spot PCR <.3, there were42 cases with spot PCR range of .3 to .9, there were 55 cases with spot PCR range of 1 to 1.5, there were 26 cases with spot PCR range of 1.6 to2.5 and there were16 cases with spot PCR >2.5

BAR DIAGRAM OF CASE DISTRIBUTION OF URINE ALBUMINURIA IN RELATION WITH SPOT PCR



This bar chart shows the number of cases distribution with the urine albumin frequency with spot PCR. There were 49 case with urine albumin 1+,there were 67 cases with urine albumin 2+ there were 28 case with 3+ urine albumin, and there were 6 cases with 4+ urine albumin. This shows that most of cases were in the mild proteinuria range in this study

PIE CHART OF NO. OF CASES IN RELATION TO URINE ALBUMINURIA



This is the pie chart showing the distribution of one hundred and fifty cases with the urine albuminuria distribution Most of the cases were in the mild protienuria range in this case study.

TABLE XII CASE DISTRIBUTION OF URINE ALBUMINURIA WITH 24 HOUR URINE PROTEIN RATIO

S.NO	URINE ALBUMIN	24 HOUR URINE PROTEIN RATIO					Percentage
		<300	300-1000	1001-2000	2001-3000	>3000	
1	1	13	30	1	0	0	29.3
2	2	0	9	57	4	2	48
3	3	1	0	2	15	8	17.3
4	4	0	0	0	3	5	5.3
	TOTAL	**14**	**39**	**60**	**22**	**15**	**100**

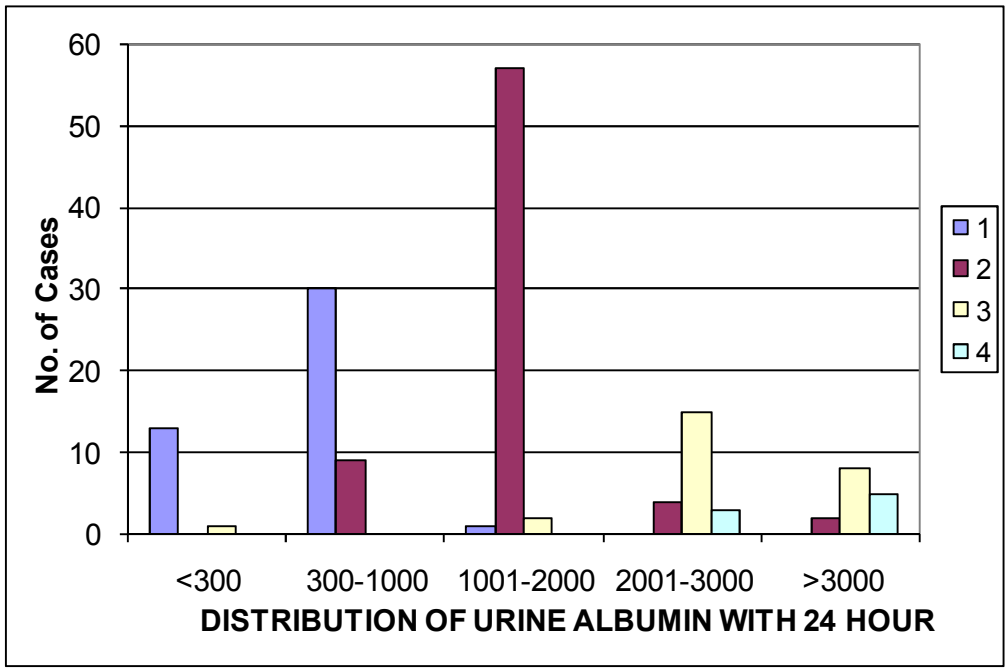
In the one hundred and fifty cases studied 29.3 % of cases were having urine albumin of 1+ in relation with 24 HUP.

48% of cases were having urine albuminuria of 2+.

17.3 % of cases were having urine albuminuria of 3+.

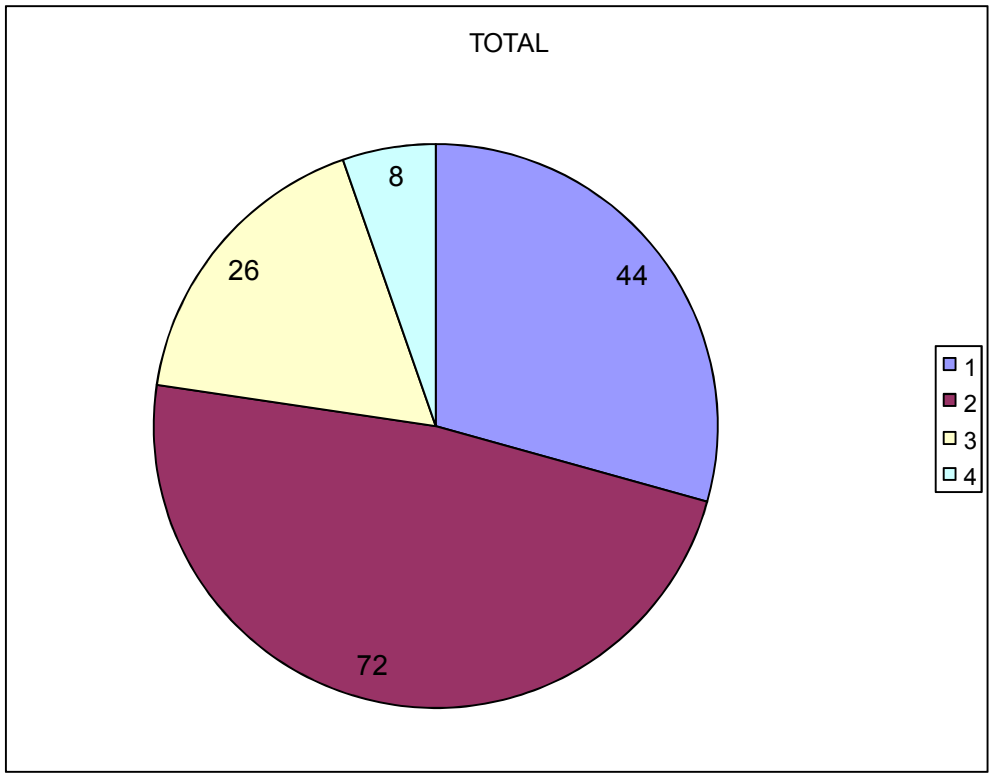
5.3% of cases were having urine albumin of 4+.

BAR DIAGRAM OF CASE DISTRIBUTION OF URINE ALBUMINURIA WITH 24 HOUR URINE PROTEIN RATIO



This bar chart shows the number of case distribution of urine albumin range frequency with the 24 hour urine protein range. There were 17 cases with urine albumin 1+ and 24 HUP <300mg/day, there were 72 cases with urine albumin 2+ with 24 HUP range between 300 to1000mg/day, there were 37 cases with urine albumin 3+with 24 HUP range ,and there were 8 cases with urine albumin 4+ with 24 HUP range >3000mg/day. This shows that most of the case were in mild degree of proteinuria, when compared to the severe degree of proteinuria.

PIE CHART OF CASE DISTRIBUTION OF URINE ALBUMINURIA WITH 24 HOUR URINE PROTEIN RATIO



This pie chart represents the distribution of number of cases with the urine proteinuria ranges.

TABLE XIII CASE DISTRIBUTION OF SPOT PCR WITH 24 HOUR URINE PROTEIN RATIO

S.NO	24 HOUR PROTEIN RATIO	SPOT URINE PCR					Total	Percentage
		<.3	.3-.9	1.0-1.5	1.6-2.5	>2.5		
1	<300	0	14	0	0	0	14	9.3
2	300-1000	11	26	7	0	0	44	29.3
3	1001-2000	0	2	47	6	0	55	36.6
4	2001-3000	0	0	0	19	3	22	14.6
5	>3000	0	0	1	1	13	15	10
	TOTAL	**11**	**42**	**55**	**26**	**16**	**150**	**100**

This table shows the relation of spot PCR with 24 hour urine protein ratio. In one hundred and fifty case studied 9.3% of cases were having spot PCR of .3,and 24 HUP of <300 mg/day.

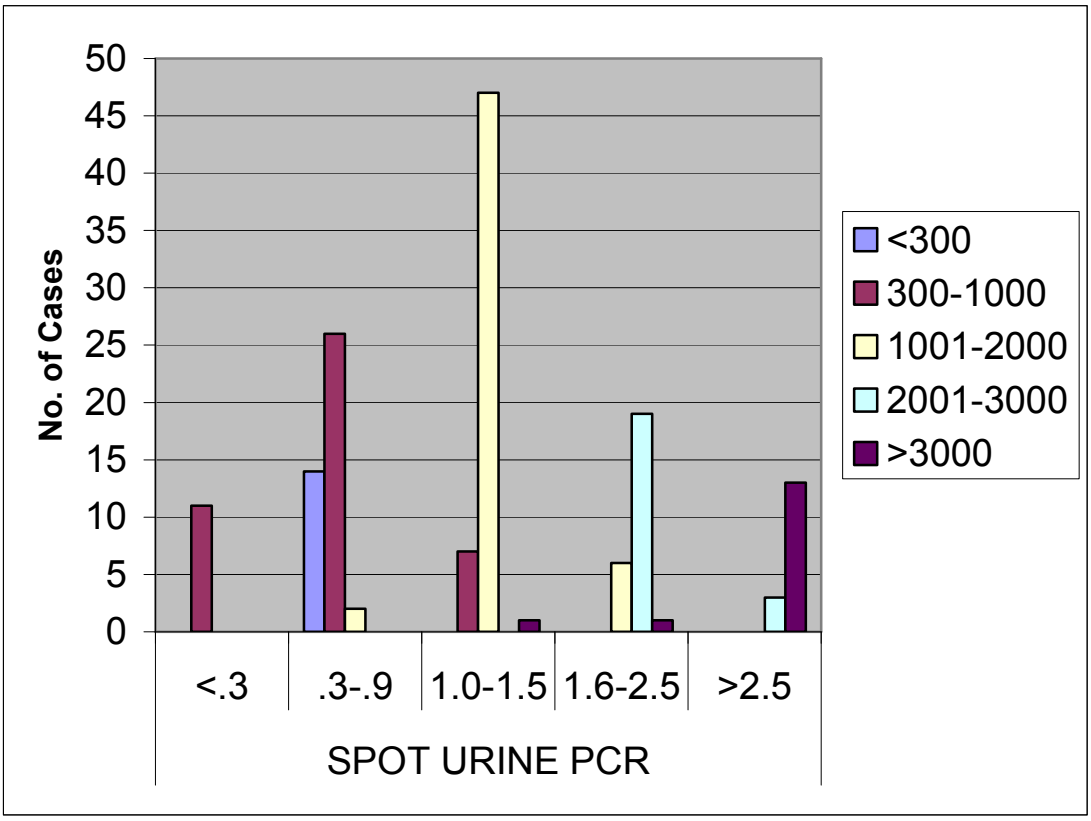
29.3 % of cases were having spot PCR of .3-.9,and 24 HUP of 300-1000mg/day.

In this study there were 36.6% of cases with spot PCR of 1-1.5 , and 24 HUP of 1001-2000mg/day.

14.6 of cases were having spot PCR of 1.6-2.5 and 24HUP of 2001-3000mg/day.

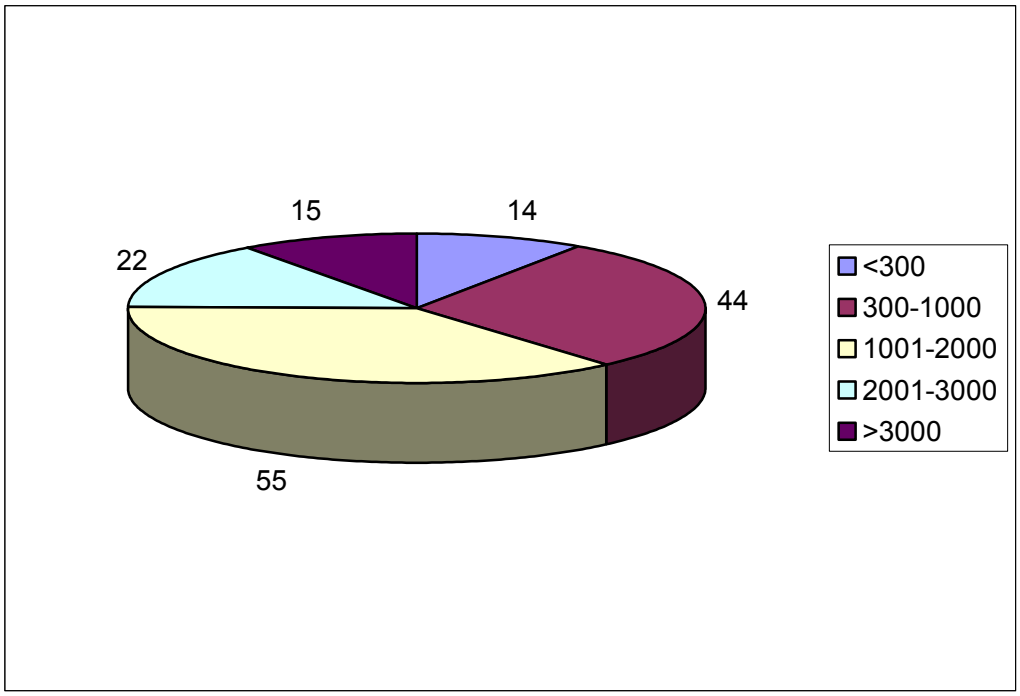
10 % of cases were having spot PCR OF >2.5 , and 24 HUP of >3000mg/day.

BAR DIAGRAM OF CASE DISTRIBUTION OF SPOT PCR WITH 24 HOUR URINE PROTEIN RATIO



In this case study there were 11 cases with spot PCR <.3, and 14 cases with 24 HUP <300 3g/day. There were 42 cases with spot PCR range of .3 - .9 44 cases with 24 HUP of range 300 – 1000 mg/day. There were 55 cases with spot PCR 1 – 1.5 range, and 55 cases with 24 HUP of 1001 – 2000 mg/day. There were 26 cases with spot PCR 1.6 – 2.5 and 22 cases with 24 HUP of 2001 – 3000 mg/day. There were 16 cases with spot PCR > 2.5 & 15 cases with 24 HUP more than 3000 mg/day, which showed a significance correlation between the Spot PCR & 24 HUP.

PIE CHART OF CASE DISTRIBUTION OF 24 HOUR WITH URINE PROTEIN RATIO



PIE CHART OF CASE DISTRIBUTION OF SPOT PCR

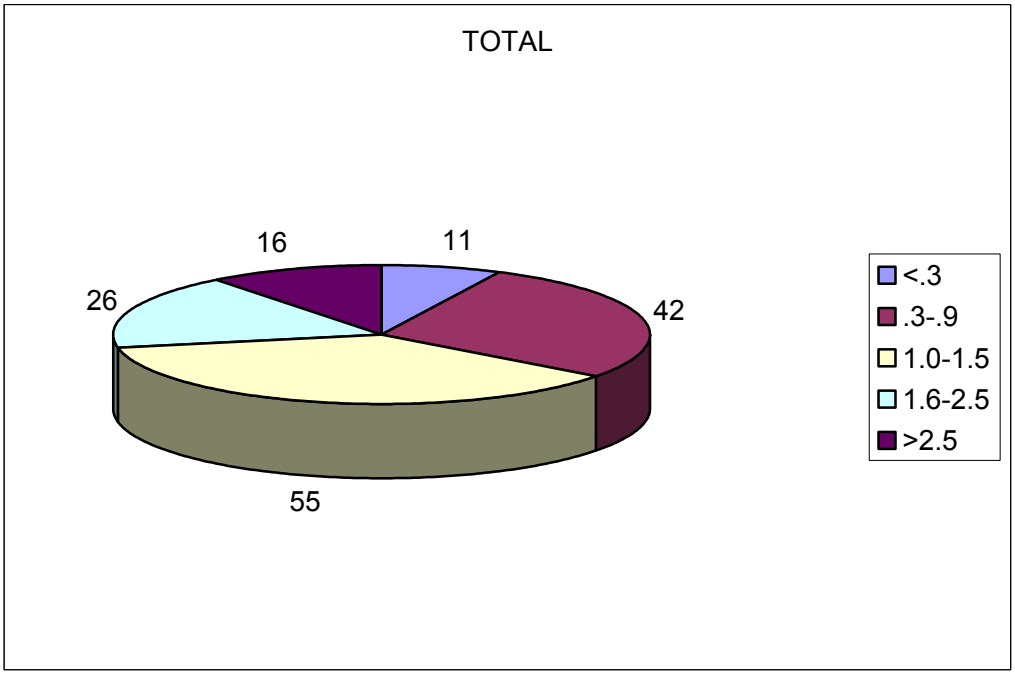
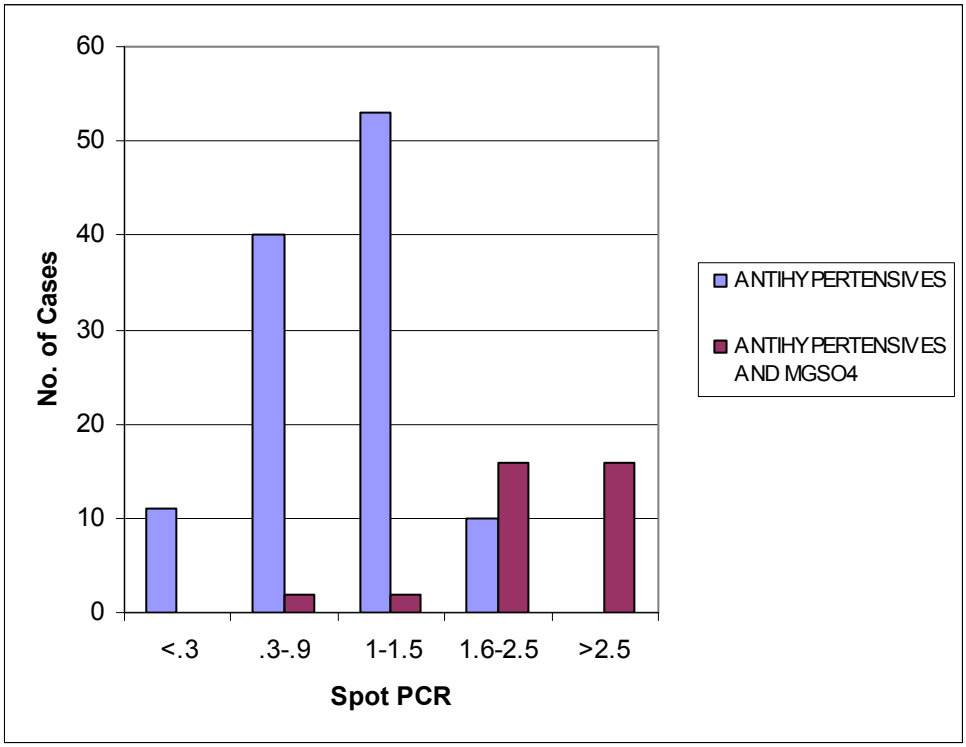


TABLE XIV DISTRIBUTION OF MANAGEMENT WITH SPOT PCR

S.NO	MANAGEMENT	SPOT PCR					Percentage
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1	ANTIHYPERTENSIVES	11	40	53	10	0	76
2	ANTIHYPERTENSIVES AND MGSO4	0	2	2	16	16	24
	Total	**11**	**42**	**55**	**26**	**16**	**100**

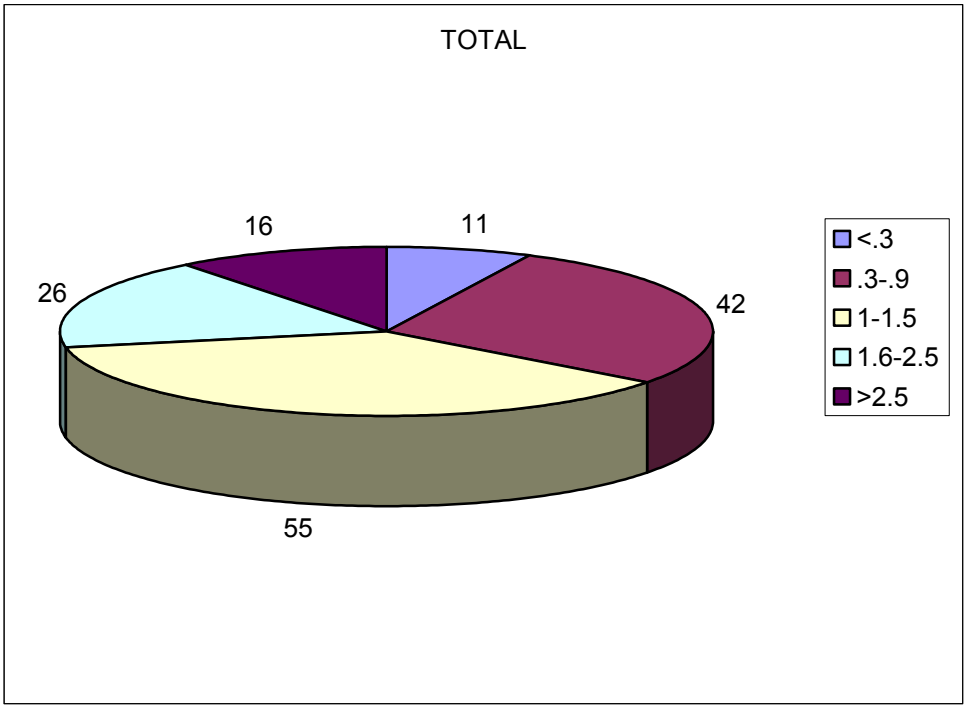
This table shows that 76% of the preeclampsia cases were managed with anti hypertensives drugs that included Labetolol and Nifedipine in the antenatal period and Atenolol in the postpartum period.

24% of cases were managed with MGSO4 regimen along with antihypertensives.



This bar chart represents the case distribution of spot PCR and the management of the cases with anti. hypertensives and MGSO4 and the frequency distribution of cases with the spot PCR. There were 11 cases with spot PCR <.3,therewere 42 cases with spot PCR .3 TO .9 ,there were 55 cases with spot PCR 1 to 1.5,there were 26 cases with spot PCR 1.6 to2.5, and there were 16 cases with spot >2.5 in this study .This shows that as the severity of the spot PCR increases, the cases were managed with antihypertensives and MGSO4.

PIE CHART OF CASE DISTRIBUTION FOR MANAGEMENT IN RELATION WITH SPOT PCR



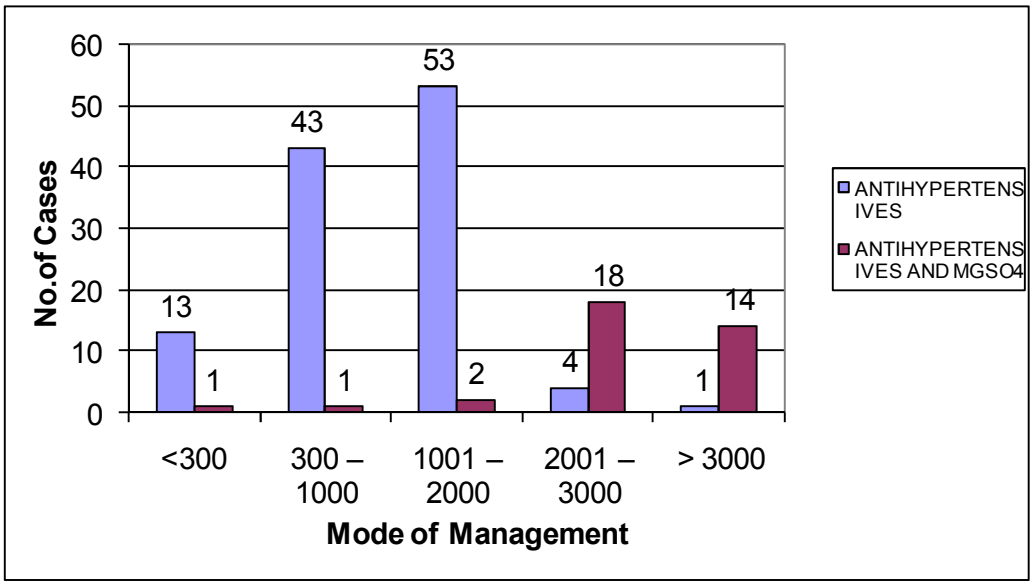
This pie chart represents the number of case distribution with spot PCR ranges.

TABLE XV DISTRIBUTION OF MANAGEMENT WITH 24 HOUR URINE PROTEINURIA

S. No	MANAGEMENT	24 HOUR URINE PROTEIN RATIO IN mg / day					Percentage
		<300	300 – 1000	1001 – 2000	2001 – 3000	> 3000	
1	ANTIHYPERTENSIVES	13	43	53	4	1	76
2	ANTIHYPERTENSIVES AND MGSO4	1	1	2	18	14	24
	Total	**14**	**44**	**55**	**22**	**15**	**100**

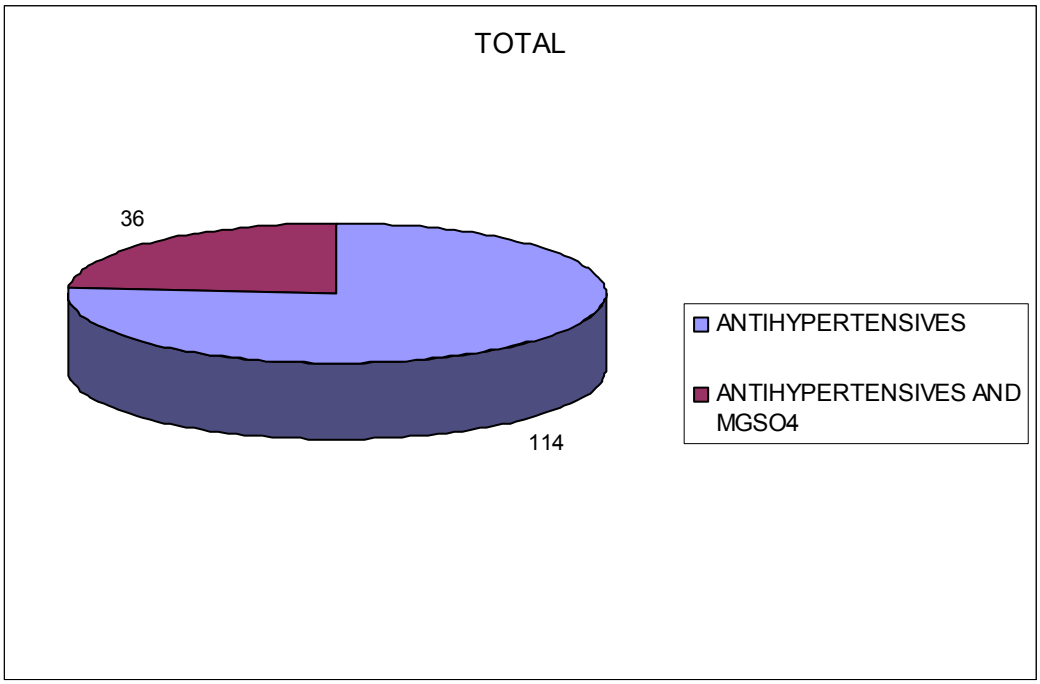
This table represents the distribution of number of cases with the 24 HUP and there management with the antihypertensives and MGSO4 regimen. This shows that as the ranges of spot PCR increases the cases were managed with both antihypertensives and MGSO4 regimen.

BAR DIAGRAM OF MANAGEMENT WITH 24 HOUR URINE PROTEINURIA



This bar chart represents the distribution of number of cases with spot PCR ranges and the management of the one hundred and fifty cases with antihypertensives and MGSO4 REGIMEN

PIE CHART OF MANAGEMENT WITH 24 HOUR URINE PROTEINURIA



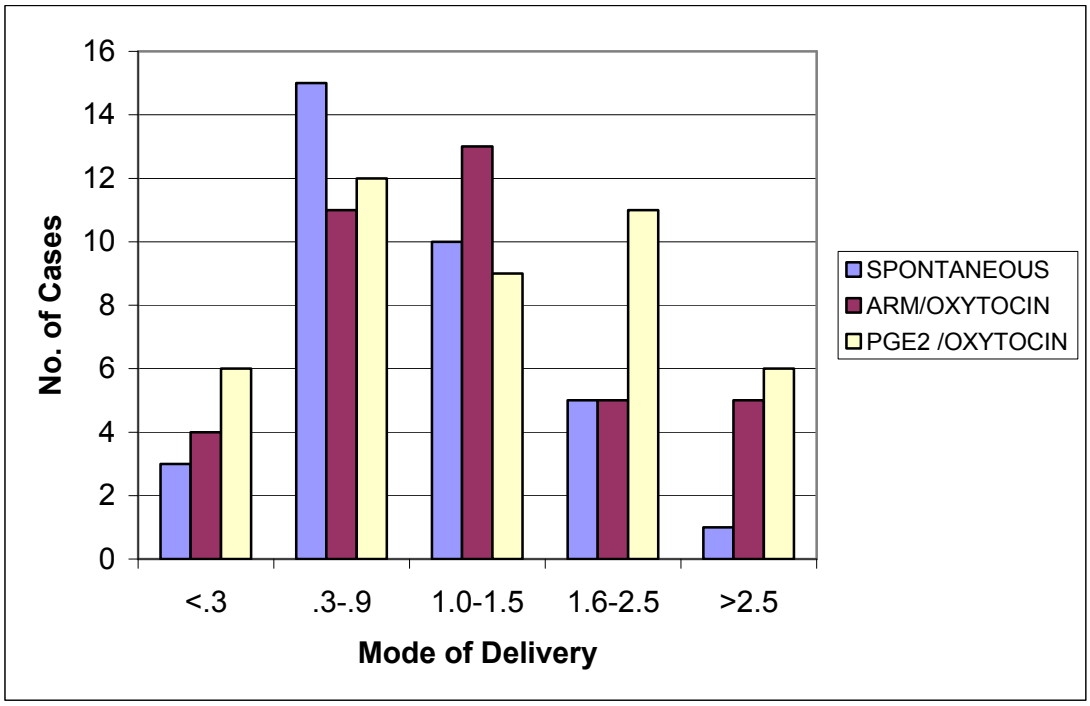
This pie chart represesnts number of cases managed with the anti hypertensives and MGSO4 regimen.

TABLE XVI MODE OF DELIVERY WITH SPOT PCR

S. NO	MODE OF DELIVERY	SPOT PCR					Total
		<.3	.3-.9	1.0-1.5	1.6-2.5	>2.5	
1	SPONTANEOUS	2	11	20	3	1	37
2	ARM/OXYTOCIN	3	16	11	3	3	38
3	PGE2 /OXYTOCIN	6	9	13	8	5	41

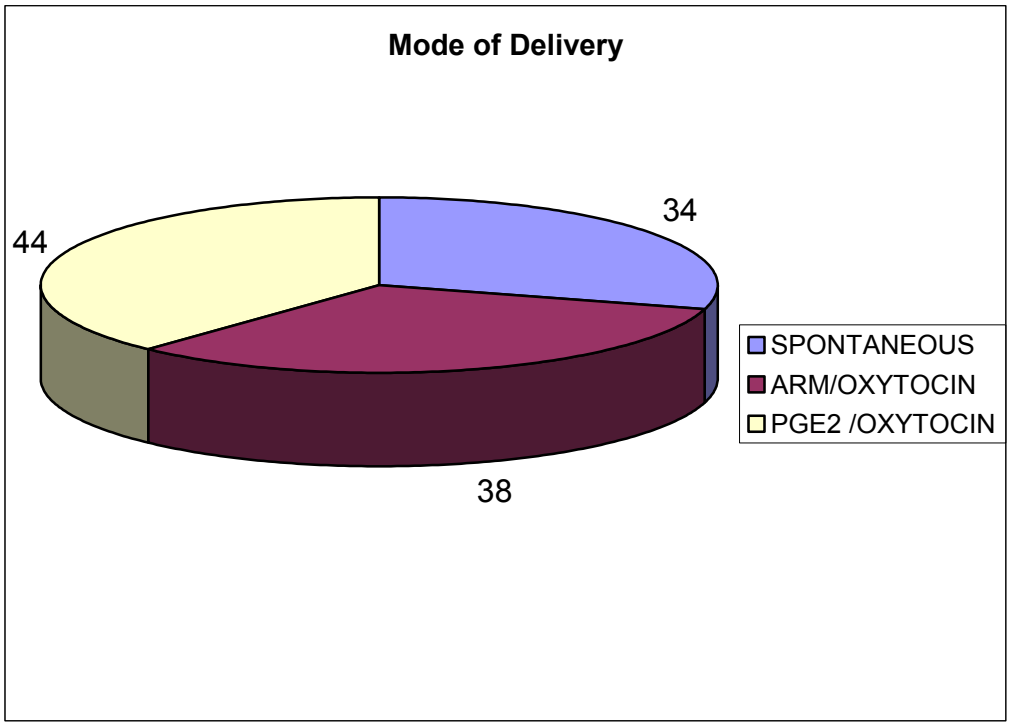
This table shows the mode of delivery of 150 cases. The mode of delivery depended upon the blood pressure, degree of proteinuria, the gestational age, maternal complication and feal complication as blood pressure and proteinuria increased, the cases were decided accordingly. In this case study, 37 cases went in for spontaneous onset of labour, for 38 cases acceleration of labour was done with ARM / OXYTOCIN, for 41 cases labour was induced with PGE2 gel and Oxytocin. 34 cases were previous LSCS which were taken for repeat section.

BAR DIAGRAM OF DISTRIBUTION OF MODE OF DELIVERY WITH SPOT PCR



This bar chart shows the distribution of delivery with spot PCR .This shows ,as the spot PCR increased the number of cases with spontaneous onset of labour,induction were decreased.

PIE CHART FOR THE DISTRIBUTION OF MODE OF DELIVERY IN RELATION TO SPOT PCR



This is the pie chart distribution of the one hundred and fifty cases and there mode of delivery in relation to spot PCR

TABLE XVII DISTRIBUTION OF MODE OF DELIVERY WITH 24 HOUR URINE PROETIN RATIO

S. NO	MODE OF DELIVERY	24 HOUR URINE PROTEIN RATIO					TOTAL
		<300	300-1000	1001-2000	2001-3000	>3001	
1	SPONTANEOUS	1	15	18	2	1	**37**
2	ARM/OXYTOCIN	9	12	14	3	4	**36**
3	PGE2/OXYTOCIN	4	13	14	7	5	**43**

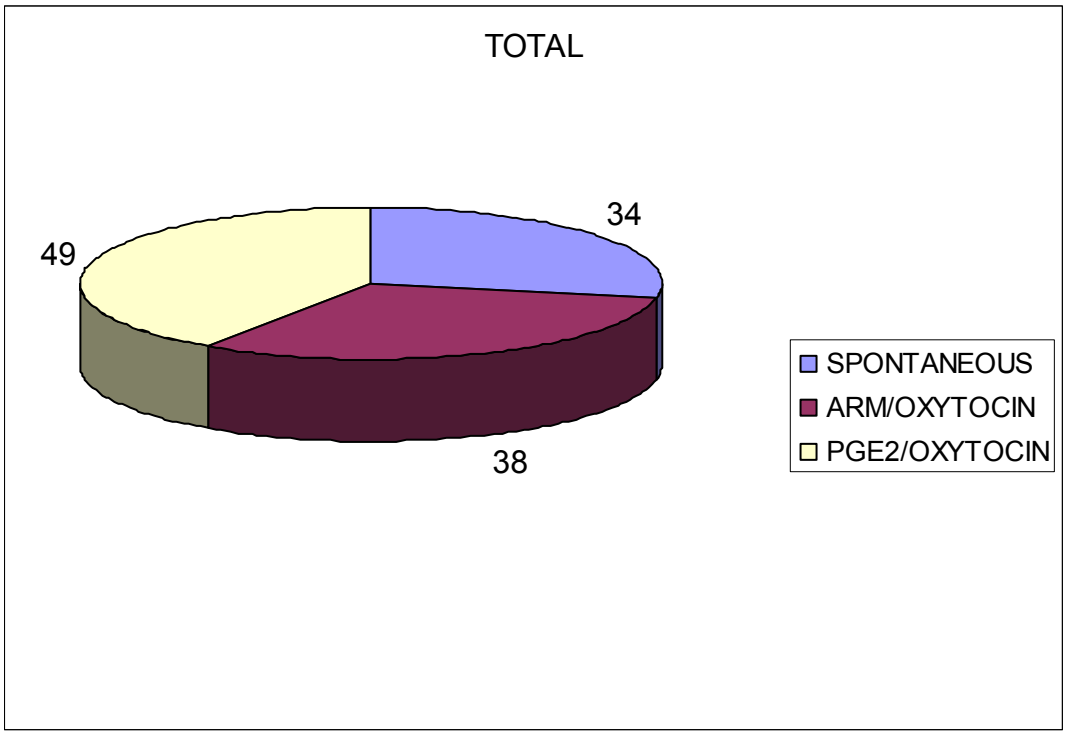
This table shows the distribution of mode of delivery with the 24 HUP ranges. As the 24 HUP range increased the cases were decided upon the fetal and the maternal outcome and emergency LSCS were done.

This table shows the mode of delivery of 150 cases. The mode of delivery depended upon the blood pressure, degree of proteinuria, the gestational age, maternal complication and feal complication as blood pressure and proleninuria increased, the cases were decided accordingly. In this case study, 37 cases went in for spontaneous onset of labour, for 36 cases acceleration of labour was done with ARM / OXYTOCIN, for 43 cases labour was induced with PGE2 gel and Oxytocin . 34 cases were previous LSCS which were taken for repeat section.

BAR DIAGRAM OF DISTRIBUTION OF MODE OF DELIVERY WITH 24 HOUR URINE PROTEIN

This bar chart depicts the number of cases and the mode of delivery with the varing ranges of proteinuria. As the degree of proteinuria increased the decision delivery were taken into account depending upon the maternal and fetal complication.

PIE CHART OF DISTRIBUTION OF MODE OF DELIVERY WITH 24 HOUR URINE PROTEIN



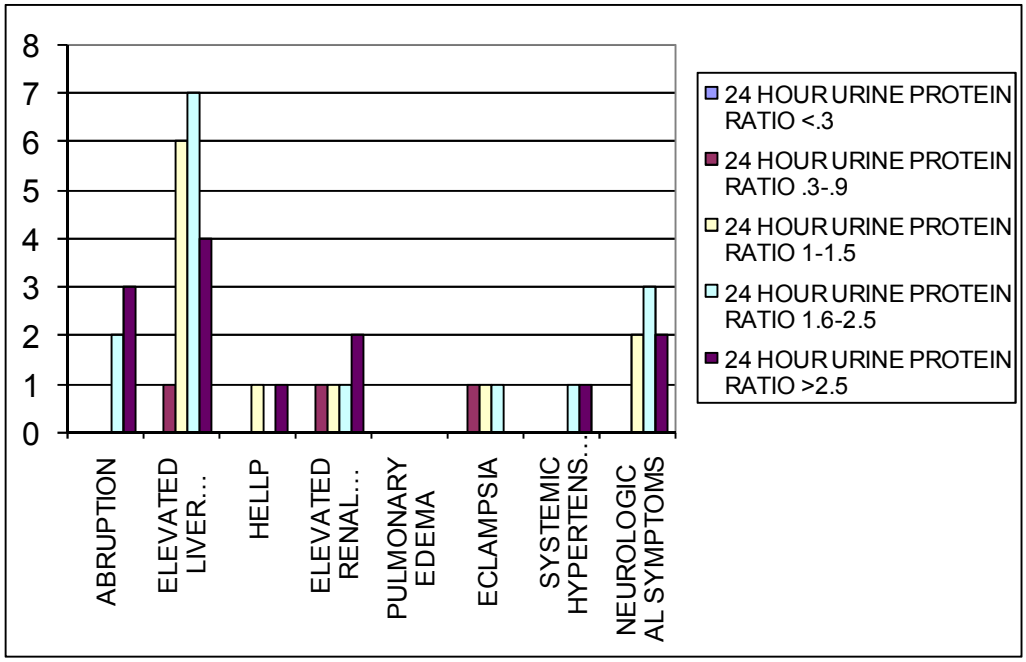
This is the pie chart distribution of mode of delivery and number of cases in relation to 24 hour urine proteinuria

TABLE XVIII DISTRIBUTION OF MATERNAL OUTCOME WITH SPOT PCR

S.NO	MATERNAL OUTCOME	24 HOUR URINE PROTEIN RATIO					Total
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1	ABRUPTION	0	0	0	2	3	5
2	ELEVATED LIVER ENZYMES	0	1	6	7	4	18
3	HELLP	0	0	1	0	1	2
4	ELEVATED RENAL VALUES	0	1	1	1	2	5
5	PULMONARY EDEMA	0	0	0	2	3	5
6	ECLAMPSIA	0	1	1	1	0	3
7	SYSTEMIC HYPERTENSION	0	0	0	1	1	2
8	NEUROLOGICAL SYMPTOMS	0	0	2	3	2	7

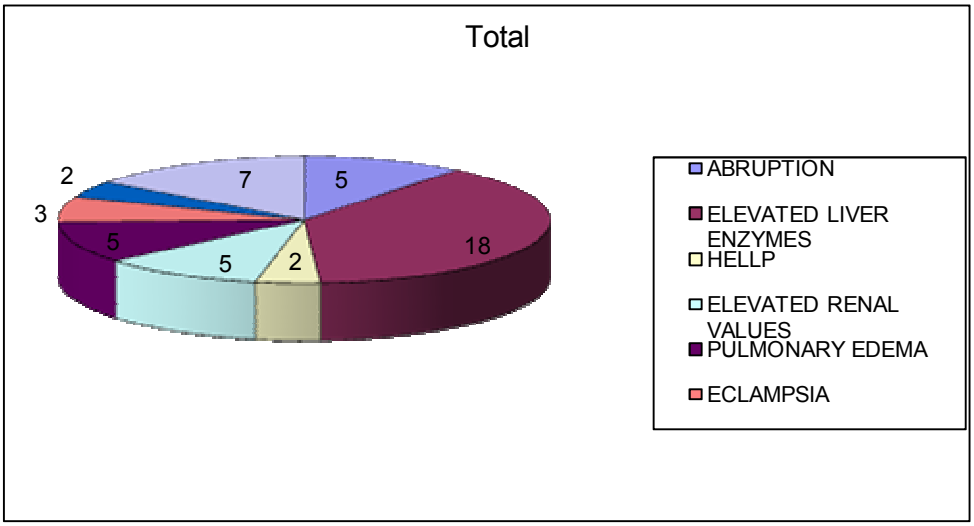
This table represents the number of cases and the adverse maternal outcome in relation with the varying spot PCR ranges. As the spot PCR increases the adverse maternal outcome increases.

BAR DIAGRAM OF DISTRIBUTION OF MATERNAL OUTCOME WITH SPOT PCR



This bar chart represents thenumber of cases of adverse maternal outcome n relation to the increasing spot urine PCR.

PIE CHART OF DISTRIBUTION OF MATERNAL OUTCOME WITH SPOT PCR



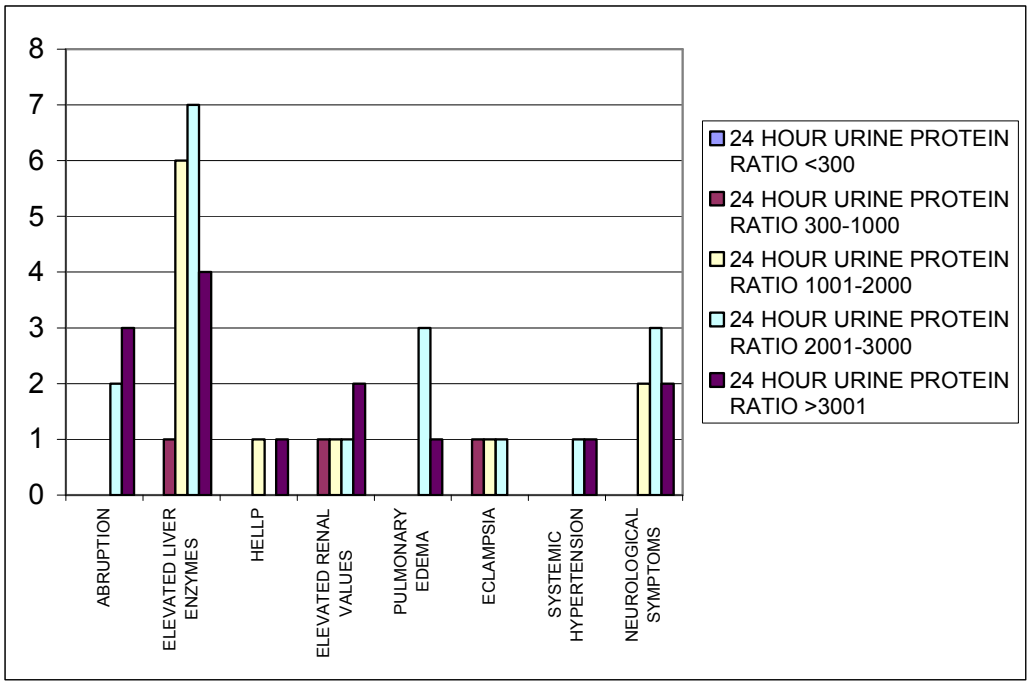
This pie chart represents number of adverse maternal outcome in relation to the spot PCR. As the spot PCR increased there was a strong association of adverse maternal outcome.

TABLE XIX DISTRIBUTION OF MATERNAL OUTCOME WITH 24 HOUR URINE PROTEIN RATIO

S.NO	MATERNAL OUTCOME	24 HOUR URINE PROTEIN RATIO					TOTAL
		<300	300-1000	1001-2000	2001-3000	>3001	
1	ABRUPTION	0	0	0	2	3	5
2	ELEVATED LIVER ENZYMES	0	1	6	7	4	18
3	HELLP	0	0	1	0	1	2
4	ELEVATED RENAL VALUES	0	1	1	1	2	5
5	PULMONARY EDEMA	0	0	0	3	1	4
6	ECLAMPSIA	0	1	1	1	0	3
7	SYSTEMIC HYPERTENSION	0	0	0	1	1	2
8	NEUROLOGICAL SYMPTOMS	0	0	2	3	2	7

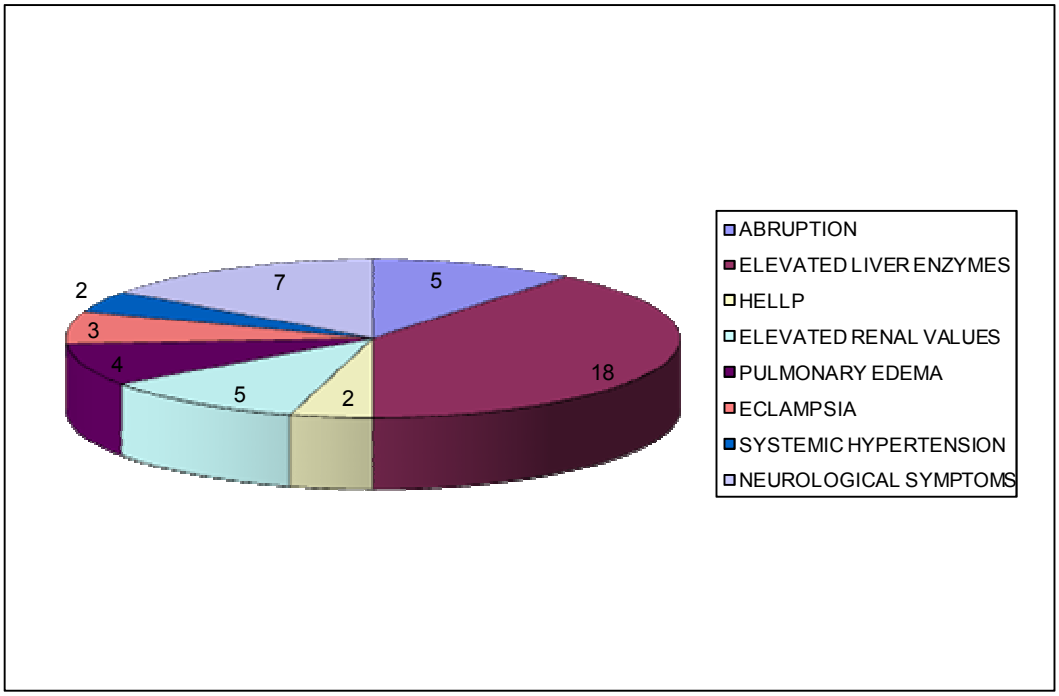
This table represents the maternal outcome depending up on the 24 HUP ranges. As the 24 HUP range increased the adverse maternal outcome also increased

BAR DIAGRAM OF DISTRIBUTION OF MATERNAL OUTCOME WITH 24 HOUR URINE PROTEIN RATIO



This bar chart shows the distribution of maternal outcome in relation to 24 hour urine protein ratio. As the 24 hour urine proteinuria increased the adverse maternal outcome also increased.

THIS PIE CHART REPRESENTS THE NUMBER OF CASES AND THE MATERNAL ADVERSE OUTCOME.



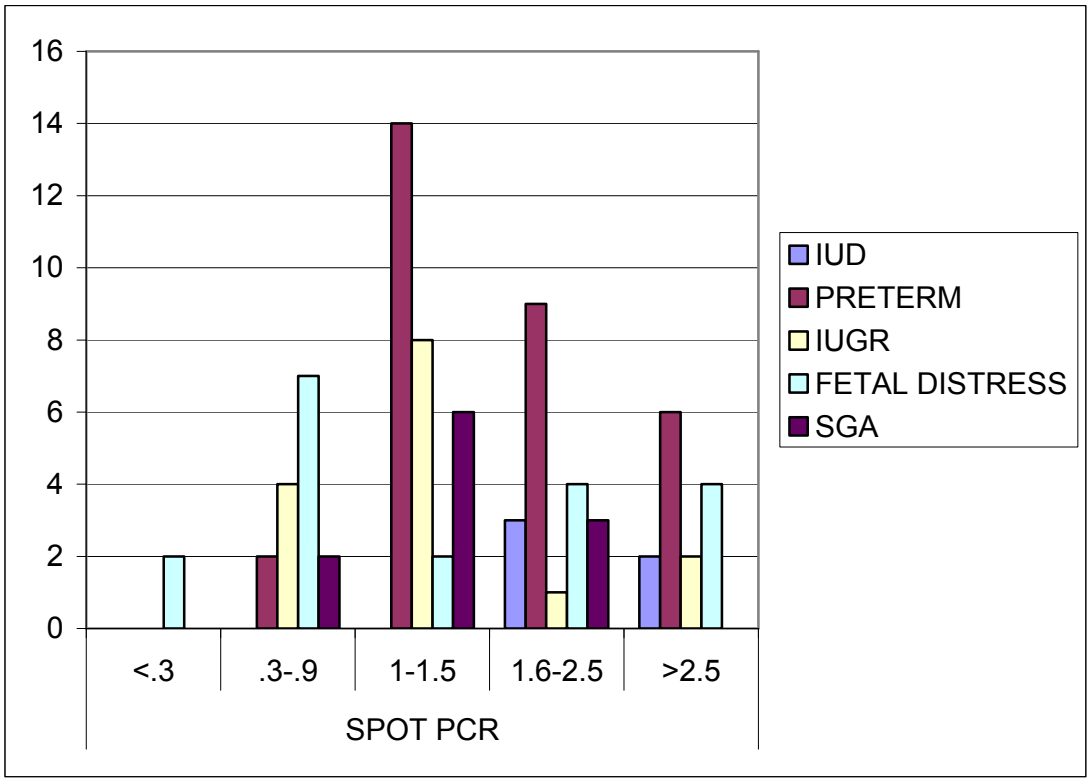
This pie chart shows the distribution of maternal outcome and number of case distribution with 24 hour urine protein ratio

TABLE XX DISTRIBUTION OF FETAL OUTCOME WITH SPOT PCR

S.NO	FETAL OUTCOME	SPOT PCR					TOTAL
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1	IUD	0	0	0	3	2	5
2	PRETERM	0	2	14	9	6	31
3	IUGR	0	4	8	1	2	15
4	FETAL DISTRESS	2	7	2	4	4	9
5	SGA	0	2	6	3	0	11

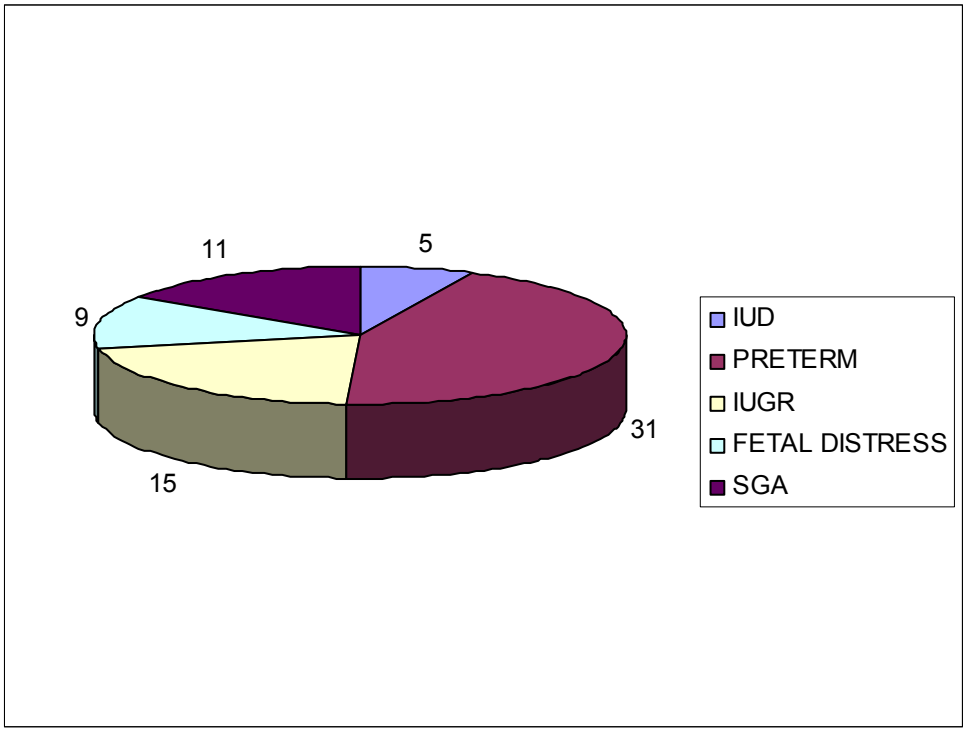
This table shows the fetal adverse outcome in relation to the varying ranges of the spot PCR. As the spot PCR increased the adverse fetal outcome also increased.

BAR DIAGRAM OF FETAL OUTCOME WITH SPOT PCR



This bar chart diagram represents the fetal outcome and the number of cases in relation to the spot PCR.

PIE CHART OF FETAL OUTCOME WITH SPOT PCR



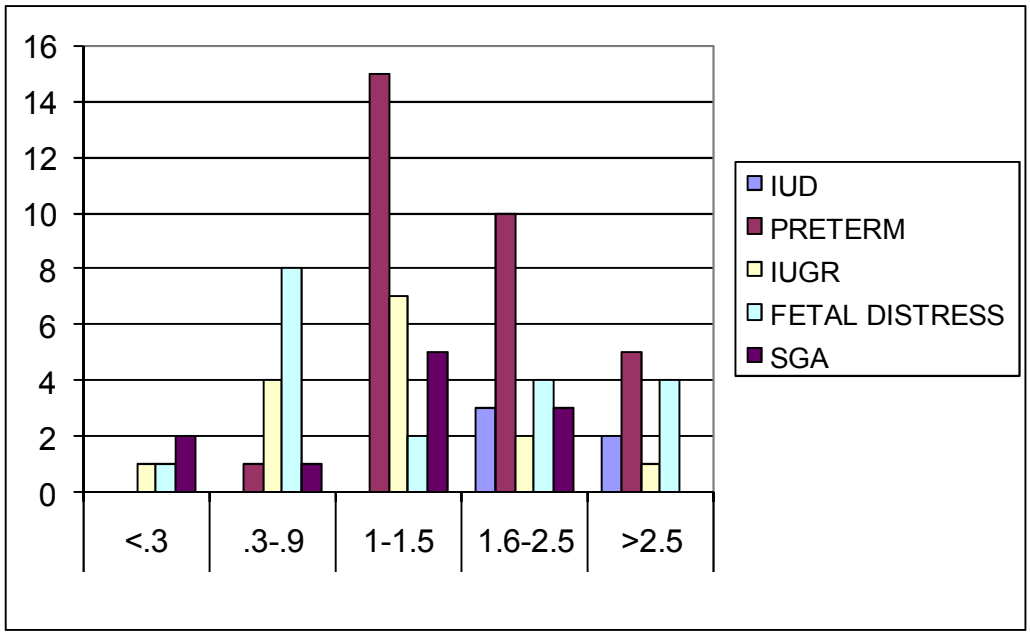
This pie chart represents the total number of cases and the adverse fetal outcome in this study.

TABLE XXI DISTRIBUTION OF FETAL OUTCOME WITH 24 HOUR URINE PROTEIN RATIO

S.NO	FETAL OUTCOME	24 HOUR URINE PROTEIN RATIO					TOTAL
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1	IUD	0	0	0	3	2	5
2	PRETERM	0	1	15	10	5	31
3	IUGR	1	4	7	2	1	15
4	FETAL DISTRESS	1	8	2	4	4	19
5	SGA	2	1	5	3	0	11

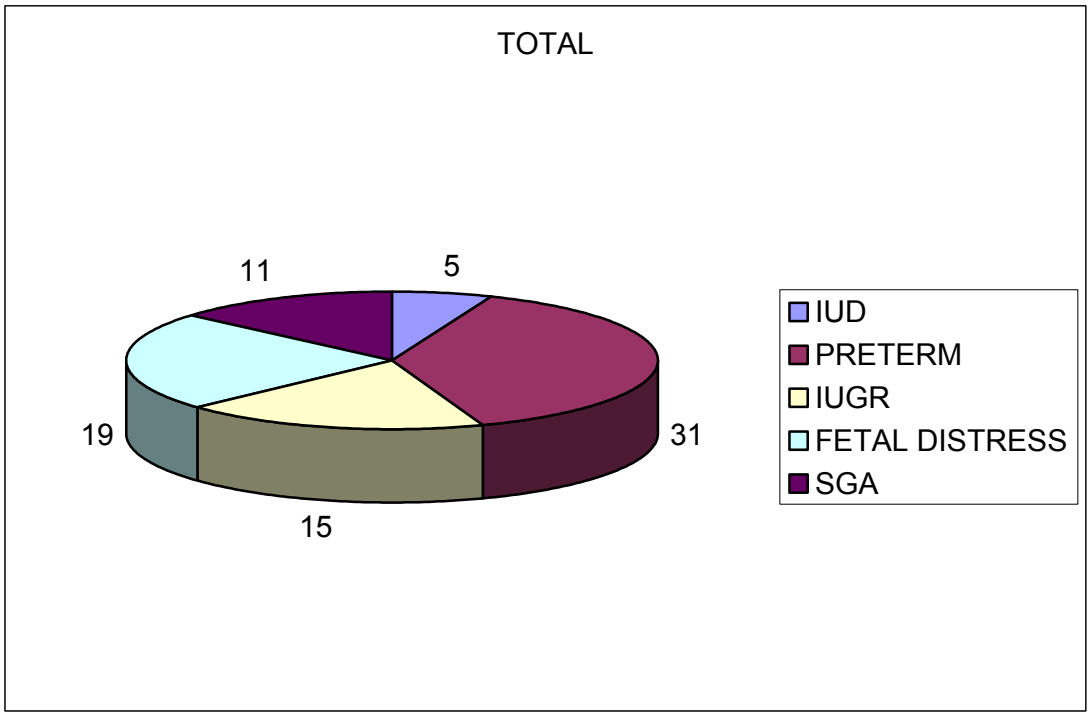
This table represents the adverse fetal outcome with the varying ranges of 24 HUP. As the 24 HUP increased the fetal mortality and morbidity were increased.

BAR DIAGRAM OF DISTRIBUTION OFFETAL OUTCOME WITH 24 HOUR URINE PROTEIN



This bar chart represents the distribution of number of cases with the varing ranges of 24 HUP.

PIE CHART OF DISTRIBUTION OFFETAL OUTCOME WITH 24 HOUR URINE PROTEIN



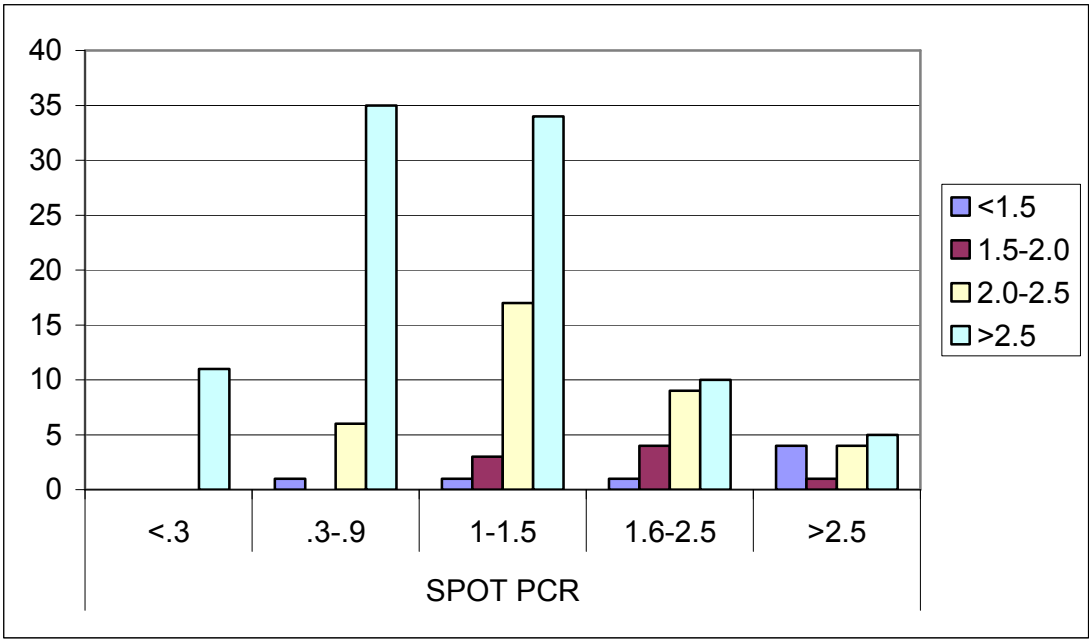
This Pie Chart represents the number of cases and fetal morbidity and the fetal outcome.

TABLE XXII DISTRIBUTION OF BIRTH WEIGHT WITH SPOT PCR

S.NO	BIRTH WEIGHT IN KG	SPOT PCR					TOTAL
		<.3	.3-.9	1-1.5	1.6-2.5	>2.5	
1	<1.5	0	1	1	1	4	7
2	1.5-2.0	0	0	3	4	1	8
3	2.0-2.5	0	6	17	9	4	38
4	>2.5	11	35	34	10	5	95

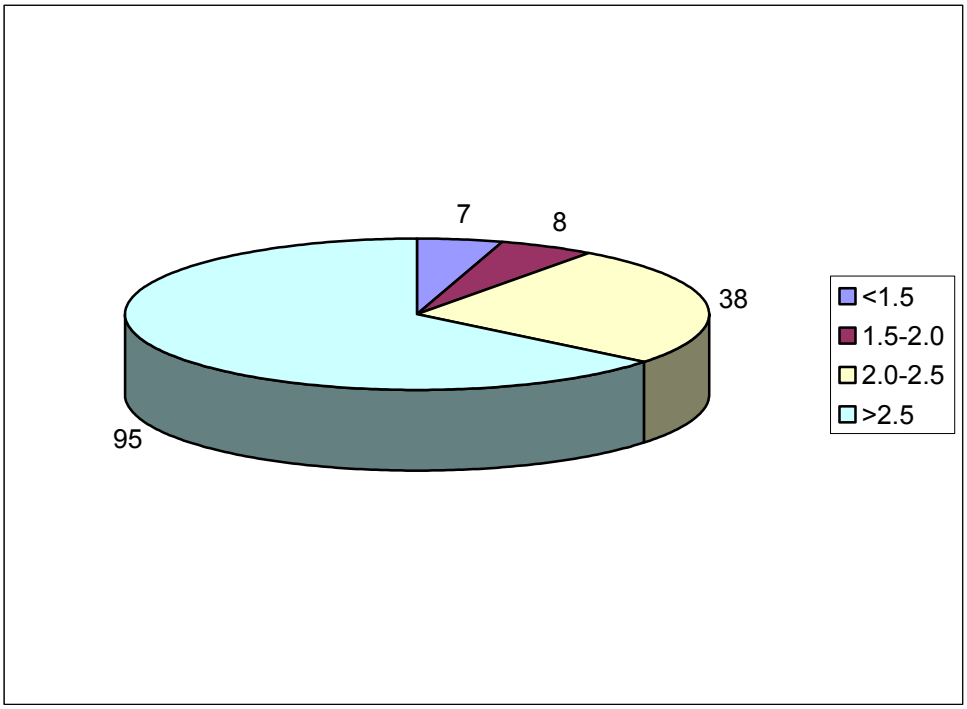
This table shows the distribution of number of cases with varying ranges of spot PCR, and birth weight of one hundred and fifty cases studied.

BAR DIAGRAM OF DISTRIBUTIONOF BIRTH WEIGHT WITH SPOT PCR



This bar diagram represents the number of cases and the birth weight in relation to the varying ranges of spot PCR.

PIE CHART OF DISTRIBUTIONOF BIRTH WEIGHT WITH SPOT PCR



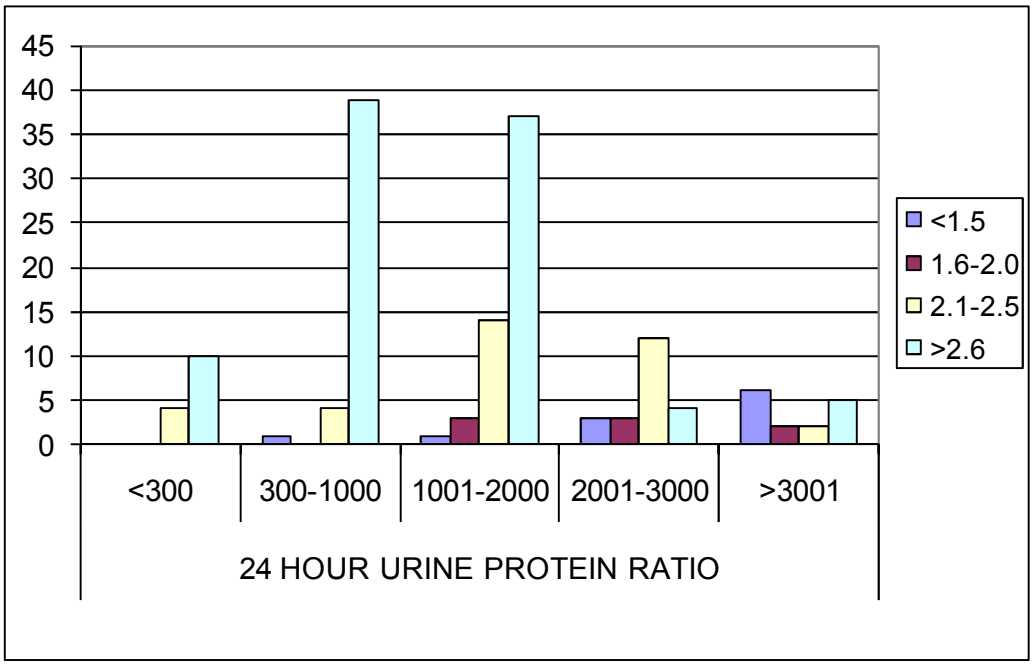
This pie chart represents the number of cases and the birth weight in relation with spot PCR.

TABLE XXIII DISTRIBUTION OF BIRTH WEIGHT WITH 24 HOUR URINE PROTEIN RATIO

S.NO	BIRTH WEIGHT	24 HOUR URINE PROTEIN RATIO					TOTAL
		<300	300-1000	1001-2000	2001-3000	>3001	
1	<1.5	0	1	1	3	6	11
2	1.6-2.0	0	0	3	3	2	8
3	2.1-2.5	4	4	14	12	2	36
4	>2.6	10	39	37	4	5	95

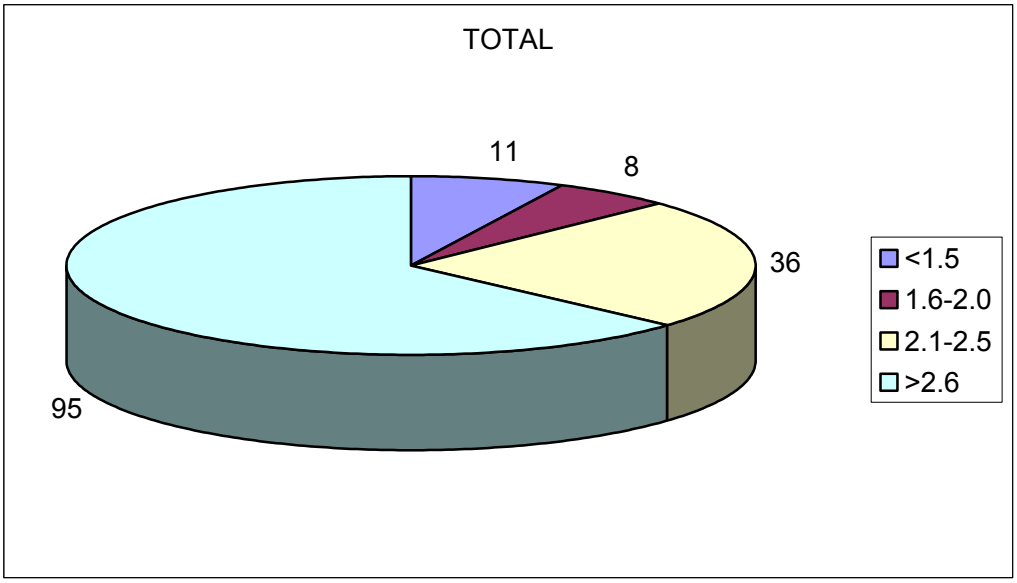
This table represents the number of cases and the fetal birth weight and the varying range of 24 HUP.

BAR DIAGRAM OF DISTRIBUTION OF BIRTH WEIGHT WITH 24 HOUR URINE PROTEIN RATIO



This bar diagram represents the birth weight distribution and the number of cases and the varying degrees of 24 HUP.

PIE CHART OF DISTRIBUTION OF BIRTH WEIGHT WITH 24 HOUR URINE PROTEIN RATIO



This pie chart represents the birth weight distribution and the number of cases in relation with the varying degrees 24 HUP.

DISCUSSION AND SUMMARY

This study was carried on a 150 selected admitted antenatal cases with the preeclampsia complicating pregnancy with gestational age >20 wks. Women with urinary tract infection ,renal disorder were excluded.

The spot urine protein creatinine ratio sample was collected before the collection of 24 hour urine sample in a clean test tube.

24 hour urine collection was started after voiding the early morning urine. The time was noted. The 24 hour urine was collected in a clean bottle and the last sample was taken on the next day at the same time .Urine protein level was measured in both the samples with the calorimetric method using pyrogallol red molybate compound and urine creatinine level was determined by creatinine Jaffe's method.

The ratio was obtained by dividing the urine protein in mg/mmol and urine creatinine in mg/mmol .The stastistical evaluation was done in all variables.

In this study conducted in one hundred and fifty patients of preeclampsia the results were analysed by comparing the variables like systolic blood pressure, diastolic blood pressure,urine albuminuria by dipstick method,the management of the cases by antihypertensives and

MGSO4 regimen ,the mode of delivery ,the maternal outcome,fetal outcome and birth weight results were analysed between the spot PCR and 24 hour urine protein ratio.

In this study of 150 cases of preeclampsia , 77.3% of cases were grouped under mild preeclampsia and 12.6% of cases were grouped under severe preeclampsia, depending upon the systolic blood pressure.

Based on the diastolic blood pressure there were 78.4% cases of mild preeclampsia and 14.6% of cases were categorised under severe preelampsia. Both the variables were compared, and the P value for the systolic and diastolic blood pressure for the spot PCR ,and 24 hour urine protein ratio was calculated and it was found to <0.001 which was significant.

The varying degree of urine albuminuria by dipstick method ,was compared with the spot PCR and 24 hour urine proteinuria and the P value was calculated, which was found to <0.001, which was significant .In this case study of 150 preeclampsia women there were 77.4% of cases with mild proteinuria and 22.6% of cases with severe proteinuria by the dipstick analysis.(Bhavana et al study on comarision of spot urine PCR with 24 hour urine proteinuria in preeclampsia.)

In this case study of 150 patients with preeclampsia 114 patients were managed with antihypertensives, which contributed to 76% of the total cases and 24% of cases were managed with antihypertensives and MGSO4 regimen. The P value for the management of cases between the spot PCR and 24 hour urine protein ratio was calculated which was found to be <0.001, which was significant.

When analysing the maternal outcome of the 150 case of preeclampsia ,there were 8 case of Abruption ,which was managed by stabilisation of the patients urgent investigation, immediate measure to control the hypertension and immediate termination of pregnancy and efficient postpartum management In this study there were18 cases with elevated liver enzymes like raised alkaline phosphatase, there were 5 case with raised serum uric acid values, and 3 cases with Eclampsia,7 cases with neurological symptoms like blurring of vision, in the 150 case followed postpartum 2 cases were found to have persistent hypertension which was referred the medicine department and nephrology department and the patients are continuing antihypertensives.This shows that as the spot urine PCR increased the adverse maternal outcome also increased .(Chan et al study on spot urine PCRas there is an increase in spot urine PCR there is an increasing risk of maternal adverse outcome.

The P value for the maternal outcome, between the spot PCR and 24 hour urine protein was calculated and it was found to <0.001 which was significant.

The fetal outcome for 150 cases were analysed .There were 5 cases of IUD, 31 cases of preterm,9 cases of fetal distress,11,cases of small for gestational age.

The fetal outcome P value was calculated by comparing the fetal outcome with spot PCR and 24 hour urine protein ratio which was significant, that is P =0.001,

The birth weight of 150 cases were analysed and P value of birth weight variable between spot PCR and 24 hour protein ratio was calculated which was found to be significant that is 0.001.(Morris et al study on spot urine PCR shows as the spot PCR increased the fetal adverse outcome also increased).

Coefficient correlation was used to determine the correlation between the urine protein excertion and spot urine protein creatinine ratio.

In this study there was a significant correlation between 24 hour urine protein and spot protein creatinine ratio r=.869, p<0.001.

24 hour urine protein collection was often used during pregnancy to quantify proteinuria. For years this has been the standard for the diagnosis of and treatment of preeclampsia. However, 24 hour urine collection are cumbersome, subjective to collective error, requires patient's compliance and result in a greater than 24 hour delay in diagnosis from the start of collection .We found an excellent correlation between single voided urine protein creatinine ratio and 24 hour urine protein. Reliability on a single voided spot protein creatinine ratio decreases the need for patients compliance minimizes collection and laboratory errors and saves almost a day in ascertaining the results.

This study had found close correlation $r = .869$, $p < 0.001$, between spot urine protein creatinine ratio and 24 hour urine protein excretion.

Several studies showed excellent correlation between spot protein creatinine ratio and 24 urine protein excretion. Those studies were

Studies	**No of cases**	**Correlation coffiecient**	**P value**
Rober M et al	n = 71	0.94	0.001
Rodriquez Thompson	n = 90	0.80	0.001
Sauden et al	n = 100	0.93	0.001
Neither et al	n = 30	0.94	0.001
Present study	n = 150	0.869	0.001

CONCLUSION

Since the level of urinary protein excretion has considerable clinical implication in the course of pregnancy and on the maternal and perinatal outcome the early detection of even minor degrees of proteinuria is important. Dipstick analysis and sulfo salicyclic acid method as a screening for proteinuria lacks reliability with high rate of false positives .For years 24 hour urine proteinuria has been standard for the quantification of proteinuria in the management in the management of in women with preeclmpsia. However this method is cumbersome, subjective to collection error, require good patients compliance and results in delay in more than 24 hour from the start of collection. The present study indicates the value of protein creatinine ratio the diagnosis than urine 24 hour urine protein ratio. Furthermore spot urine creatinine ratio method had been found to be far more cost effective and increased the patients compliance than a 24 hour urine protein excretion ratio .Therefore the spot protein creatinine ratio can be used as an alternative to quantify the urine protein excretion in a 24 hour collection in preeclampsia complicating antenatal women.

BIBLIOGRAPHY

1. Cunningham, Gary F., Kenneth J. Leveno, Steven L. Bloom, John C. Hauth, Dwight J. Rouse and Catherine Y. Spong. Pregnancy Hypertension. In: William's Obstetrics, 2010; 231d edn. pp. 706 - 756.

2. Walfish, Asant and Mordechai Hallak. Hypertension : High Risk Pregnancy - management options, 2006; 3rd edn. pp. 772 - 797.

3. Arias Fernando, Shirish N Daftary, Amarnath G Bhide. Hypertensive disorders in pregnancy. Practical guide to high risk pregnancy and delivery, 2008; 3rd edn. pp. 397 - 439.

4. Renu Misra. Hypertensive disorders in pregnancy. In: Ian Donald's practical obstetric problems, 2007; 6th edn. pp. 280 - 309.

5. Dutta D.C. In: Text Book of Obstetrics, New Central Book Agency, 2001, 5^{th} edn. (Ed. Hiralal Konar). pp. 234 - 254.

6. Barrilleaux, P. S. and Martin, J. N. Hypertension therapy during pregnancy. Clin. Obstet. Gynecol., 2002, 45: 22.

7. Sibai, B. M. Chronic hypertension during pregnancy. In: (Ed. Sciarra, J) Gynecology and obstetrics, 1989, J. B. Lippincott, Philadelphia, pp. 1 - 8.

8. Levine, R. J., Ewell, M.G. and Hauth, J. C. Should definition of preeclampsia include a rise in diastolic BP of 15 mm of Hg to a level of < 90 mm of Hg in association with proteinuria? Am. J. Obstet. Gynecol., 2000, 183: 787.

9. North, R. A., Taylor, R. S. and Schellenberg, J. C. Evaluation of a definition of preeclampsia. Br. J. Obstet. Gynecol., 1999, 106: 767.

10. Long, P. A. and Oats, J. N. Preeclampsia in twin pregnancy: severity and pathogenesis. Aust. NZ. J. Obstet. Gynecol., 1987, 27: 1 - 5.

11. Verwoerd, G. R., Hall, D. R., Grove, D., Maritz, J. S. and Odendaal, H. J. Primipaternity and duration of exposure to sperm antigens as a risk factor for preeclampsia. Int. J. Obstet., Aug 2002, 78(2):121 - 126.

12. Robillard, P.Y., Hulsey, T. C. and, Perianin, J. Association of pregnancy induced hypertension with duration of sexual co-habitation before conception. Lancet, 1994, 344: 33.

13. Sibai, B. M., Caritis, S., et al. Hypertensive disorders in twin versus singleton gestations. Am. J. Obstet. Gynecol., 2000, 182: 938.

14. Maxwell, C. V., Leiberman, E., Norton, M., et al. Relationship of twin zygosity and risk of preeclampsia, Am J Obstet Gynecol., 2001, 185: 819.

15. Campbell, D. M., Mac Gillivary, I. and Carr-Hill. Preeclampsia in second pregnancy. Br. J. Obstet. Gynecol. 1985, 92: 131 - 140.

16. Walker, J. J. Preeclampsia. Lancet, 2000, 356: 1260 - 1265.

17. Sibai, B. M., Ewell, M., Levine, R. J., et al. Risk factors associated with preeclampsia in healthy nulliparous women. Am. J. Obstet. Gynecol., 1997, 177:1003 - 1010.

18. Bainbridge, S. A., Sidle, E. H. and Smith, J. N. Direct placental effect of cigarette smoke protects women from preeclampsia: the specific role of carbon monoxide and antioxidant systems in the placenta. Med. Hypothesis, 2005, 64: 17.

19. Granger, J.P., Alexander, B.T., Llinas, M. T., Bennett, W. A. and Khalil, R. A. Pathophysiology of hypertension during preeclampsia, linking placental ischaemia with endothelial dysfunction. Hypertension, Sep. 2001, 38(3 pt. 2):718 - 722.

20. Robertson, W. B. Brosens, I. and Dixon, G. Uteroplacental vascular pathology. Eur. J. Obstet. Gynecol. Reprod. Bio., 1975; 5: 47.

21. Dempsey, J. C., Sorenson, T. K., Qiu, C. F., Luthy, D. A. and Williams, M. A. History of abortion and subsequent risk of preeclampsia. J. Reprod. Med., July 2003, 48(7): 509 - 514.

22. Ward, K. and Lindheimer. Genetic factors in the etiology of preeclampsia/eclampsia. Chesley's hypertensive disorders in pregnancy, 3rd edn. Elsevier, 2009: p. 51

23. Manten, G. T., Van Der Hock, Y. Y., Marko Sikkema, J., et al. The role of lipoprotein(a) in pregnancies complicated by preeclampsia. Med. Hypothesis, 2005, 64: 162.

24. Elsheik, A., Creatsas, G., Mastorakos, G., et al. The rennin aldosterone system during normal and hypertensive pregnancy. Arch. Gynecol. Obstet., 2001; 264: 182.

25. Gant, N. F., Chand, S., Worley, R. J., Whalley, P. J., Corsby, U. P. and Mac Donald, .P C. A clinical test useful for predicting acute hypertension in pregnancy. Am. J. Obstet. Gynecol., Sep. 1974, 120(1): 1 - 7.

26. Myatt, L., Brewer, A.S., Langdon, G. et al. Attenuation of the vasoconstrictor effects of thromboxane and endothelin by nitric oxide in the human fetal placental circulation. Am. J. Obstet. Gynecol., 1992, 166: 224.

27. Taylor, R. N and Roberts, J. M. Endothelial cell dysfunction. Chesley's hypertensive disorders in pregnancy, 2nd edn. 1999, p. 135.

28. Weinstein, L. Syndrome of hemolysis, elevated liver enzymes and low platelet count : a severe consequence of hypertension in pregnancy. Am. J. Obstet. Gynecol., 1982 , 142: 159.

29. Kenny, L. Baker P. and Cunningham, F. G. Platelets, coagulation and the liver. Chesley's hypertensive disorders in pregnancy, 3rd edn. 2009, p 335.

30. Facchinetti, F., Marozia, L. and Frusca, T. et al. Maternal thrombophilia and the risk of recurrence of preeclampsia. Am. J. Obstet. Gynecol., 2009, 200:46.e1.

31. Conard, K. P., Gaber, L. W. and Lindheimer, M. D. The kidney in normal pregnancy and preeclampsia. Chesley's hypertensive disorders in pregnancy, 3rd edn. 2009, p 297.

32. Chesley, L. C. and Williams, L. 0. Renal glomerular and tubular function in relation to the hyperuricemia in preeclampsia and eclampsia. Am. J. Obstet. Gynecol., 1945, 50: 367.

33. Taufield, P. A., Ales, K. L., Resnick, L. M. et al. Hypocalciuria in preeclampsia. N. Engl. J. Med., 1987, 316: 715.

34. Marybury, Waugh. Proteinuria in preeclampsia — just what is significant? Fetal and Maternal Medicine Review, 2004, 16: 171 - 195.

35. Higby, K., Suiter, C. R., Phelps, J.Y., Siler-Khodr, T. and Langer, 0. Normal values of urinary albumin and total protein excretion during pregnancy. Am. J. Obstet. Gynaecol. 1994; 171: 984 — 189

36. Bernard, A., Thielemans, N., Lauwerys, R., and van Lierde, M. Selective increase in the excretion of protein 1(Clara cell protein) and other low molecular weight proteins during normal pregnancy. Scand. J. Clin. Lab Invest. 1992; 52: 871 —878.

37. Davison, J. M. Renal function during normal pregnancy and the effect of renal disease and pre-eclampsia. In: (Andreucci, V.E. ed.). The kidney in pregnancy. Martinus Nijhoff, Boston, 1986: 65 — 80.

38. Page, E. W. and Christianson, R. Influence of blood pressure changes with and without proteinuria upon outcome of pregnancy. Am. J. Obstet. Gynaecol. 1976; 126: 821 — 829.

39. Chesley, L.C. Superimposed pre-eclampsia or eclampsia. In: (Chesley, L.C. ed.) Hypertensive disorders in pregnancy. Appleton-Century-Crofts, New York, 1978.

40. Sibai, B.M., Abdella, T.N. and Anderson, G.D. Pregnancy outcome in 211 patients with mild chronic hypertension. Obstet. Gynaecol., 1983; 61: 571 — 576.

41. Sibai, B.M. and Anderson, G. D. Pregnancy outcome of intensive therapy in severe hypertension in the first trimester. Obstet. Gynecol. 1986; 67: 517 — 522.

42. Ferrazzani, S., Caruso, A, De Carolis, S., Martino IV, and Mancuso, S. Proteinuria and outcome of 444 pregnancies complicated by hypertension. Am. J. Obstet. Gynecol. 1990; 162: 366 — 371.

43. Page, E.W. and Christianson, R. Influence of blood pressure changes with and without proteinuria upon outcome of pregnancy. Am. J. Obstet. Gynecol. 1976; 126: 821 — 829.

44. Chua, S. and Redman, C. W. G. Prognosis for pre-eclampsia complicated by 5 g or more of proteinuria in 24 hours. Eur. J. Obstet. Gynaecol. Reprod. Biol. 1992; 43: 9 — 12.

45. Waugh, J, Bell, S.C., Kilby, M. D., Blackwell, C.N., Seed, P., Shennan, A. H. et al. Bedside urine albumin creatinine ratio testing

in hypertensive pregnancy. Hypertens. Pregnancy 2002; 21: Supplement 1, p. 103.

46. Meyer, N. L., Mercer, B.M., Friedman, S.A. and Sibai, B. M. Urinary dipstick protein: a poor predictor of absent or severe proteinuria. Am. J. Obstet. Gynecol. 1994; 170: 137 — 141.

47. Waugh, J. J. S., Bell, S.C., Kilby, M. D., Shennan, A.H. and Halligan, A. W. F. Effect of urine concentration and biochemical assay on dipstick accuracy in hypertensive pregnancy. Hypertens. Pregnancy 2001; 20: 205 — 217.

48. Ginsberg, J. M., Chang, B. S., Matarese, R. A., Garella, S. Use of single voided urine samples to estimate quantitative proteinuria. N. Eng. J. Med. 1983; 309: 1543 — 1546.

49. Steinhauslin, F. and Wauters, J. P. Quantitation of proteinuria in kidney transplant patients: accuracy of the protein/creatinine ratio. Clin. Nephrol. 1995; 43: 110 — 115.

50. Brodby, R. A., Rohde, R. D., Zeev, S., Pohl, M.A., Bain, R. P., Lewis, E. J. The urine protein to creatinine ratio as a predictor of 24 hour urine protein excretion in Type 1 diabetic patients with nephropathy. Am. J. Kid. Dis. 1995; 26: 904 — 909.

51. Quadri, K. H. M., Bernardini, J. B. S. N., Greenberg A. et al. Assessment of renal function during pregnancy using proteinto creatinine ratio and Cockroft-Gault formula. Am. J. of Kidney Diseases, Sep. 1994, 24(3): 416 - 420.

52. Adrienne, B., Neithardt, Sharon L Dooley and Jayne Borensztajn. Prediction of 24 hour protein excretion in pregnancy with a single voided urine protein to creatinine ratio. Am. J. Obstet. Gynecol., 2002, 186: 883 - 886.

53. Leanos-Miranda et al. Protein creatinine ratio in random urine samples is a reliable marker of increased 24 hour protein excretion in hospitalised women with hypertensive disorders of pregnancy. Clinical Chemistry, 2007. 53(9): 1623 - 1628.

54. Shahbazian, N. et al. A comparison of spot urine protein creatinine ratio with 24 hour urine protein excretion in women with preeclampsia. IJKD, 2008; 2: 127 - 131.

55. Nisell, H. et al. Urine albumin creatinine ratio for the assessment of albuminuria in pregnancy hypertension. Acta obstetrician et gynecologica scandinavica, 2006, 85 (11): 1327 - 1330.

56. Papanna, R., Mann, L. K., Kouides, R. W. et al. Protein creatinine ratio in preeclampsia: a systemic review. Obstet. Gynecol., 2008, 112: 135.

57. Wheeler, T. L. 2nd, Blackhurst, D. W., Dellinger, G. H. and Ramsey, P. S. Usage of spot urine protein to creatinine ratios in the evaluation of preeclampsia. Am. J. Obstet. Gynecol., 2007; 196; 65. e1-4.

58. Cote, A. M. et al. Diagnostic accuracy of urinary spot protein creatinine ratio for proteinuria in hypertensive pregnant women. Systemic Review. BMJ, May 2008, 3(336) (7651): 968 - 969.

59. Durnwald, C. and Mercer, B. Prospective comparison of total protein creatinine ratio versus 24 hour urine protein in women with suspected preeclampsia. Am. J. Obstet. Gynecol., Sep 2004, 191(3): 1049 - 1050.

60. Yamasmit, W., Wongkitisophon, K., Charoenvidhya et al. Correlation between random urinary protein-to-creatinine ratio and quantitation of 24-hour proteinuria in preeclampsia. J. Med. Assoc. Thai, Jan 2003; 86(1): 69 - 73.

61. Robert, M., Sepadj, F., Liston, R. M. and Dooley, K. C: Random protein creatinine ratio for the quantification of proteinuria in pregnancy. Obstet. Gynecol., 1997; 104: 1159 - 1164.

62. Leo Boler, Edward, A., Zbella and Norbert Gleicher. Quantitation of proteinuria in pregnancy by the use of single voided urine samples. Obstet. Gynecol, 1987, 70: 99.

63. Rodriguez - Thompson, D., Lieberman, E. S. Use of a random urinary protein/creatinine ratio for the diagnosis of significant proteinuria during pregnancy. Am. J. Obstet. Gynecol., Oct. 2001, 185(4): 808 - 811.

64. Richard A. Young, Rebecca, J. Buchanan, Robert and A. H. Kinch. Use of the protein / creatinine ratio of a single voided urine specimen in the evaluation of suspected PIH. J. Family Practice, April 1996, 42: 385 - 389.

65. Oscar, E. Jaschevatzky, Ron. P. Rosenberg, Amos Shalit et al. Protein / creatinine ratio in random urine specimens for

quantitation of protienuria in preeclampsia. Obstet. Gynecol., 1990, 75: 604.

66. Bhavana Bansal et al. Comparison of protein creatinine ratio in single voided urine sample with 24 hours urine protein for estimation of proteinuria in pregnancy induced hypertension. J Obstet Gynecol India, 2009,59(5):424-26.

67. Brown M A , Buddle M L . Inadequacy of dipstick proteinuria in hypertensive pregnancy. Aust NZ J Obstet Gynecol , 1995 , 35:366-69.

68. TorngS, Rigatto C , Rush D N et al. Urine protein to creatinine ratio as a predictor of 24 hours urine protein excretion in renal transplant patients. Transplantation , 2001, oct 27;72(8):1453-6.

ABBREVATIONS

NHBPEP	National High Blood Pressure Education Program working group
ACOG	American College Of Obstetrics And Gynaecology
SGOT	Serum glumate oxaloacetate transaminase
SGPT	Serum glutamate pyruvate transaminase
HELLP	Hemolysis elevated liver enymes and low platelets
VEGF	Vascular endothelial growth factors
PLGF	Platelet derived growth factor
SFLT	soluble fms like tyrosine -1
M RNA	Messenger Ribo Nucleic Acid
S ENG	Soluble Endoglin
IUGR	Intra uterine growth retardation
MCA	Middle cerebral artery
PCA	Posterior cerebral artery
PRES	Posterior reversible encephalopathy
HLA-G	Human leucocyte antigen-G
HLA-C	Human leucocyte antigen-C
HLA	Human leucocyte antigen
KIR	Killer immunocyte receptors
TGF	Tumor Growth Factor
TNF	Tumor necrosis factor
ISSHP	International society of Study on Hypertension
PCR	Protein creatinine ratio
MGSO4	Magnesium sulphate
ARM	Artificial rupture of membrane
LSCS	Lower segment caesarean section
P Value	Probability values

CONSENT FORM

STUDY TITLE : STUDY ON DIAGNOSTIC VALUE OF SPOT URINE ALBUMINURIA CREATININE RATIO WITH 24 HOUR URINE ALBUMINURIA CRETININE RATIO IN WOMEN WITH PREECLAMPSIAO

PARTICIPANT NAME : **AGE:** **SEX**:

IP.NO.STUDY CENTRE

ISO KGH CHENNAI 5

I confirm that I have understood the purpose of procedure for the above study, I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the procedure, I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties of published, unless as required under the law. I agree not to restrict the use of any or results that arise from the study.

I hereby consent to participate in this study of**STUDY ON DIAGNOSTIC VALUE OF SPOT URINE ALBUMINURIA CREATININE RATIO WITH 24 HOUR URINE ALBUMINURIA CREATININE RATIO IN WOMEN WITH PREECLAMPSIA**

Signature of Investigator: Place :

Date :

Study Investigators Name Institution

Signature / Thumb Impression of patient

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. A. Chithra,
PG in Obstetrics & Gynaecology,
ISO KGH, Triplicane,
Chennai-5.

Dear Dr. A. Chithra,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Diagnostic value of Spot Urine Protein Creatinine Ratio with 24 Hrs Urine Protein Creatinine Ratio in Antenatal Women with Preeclampsia"** No.28032014

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D. Dean, MMC, Ch-3.	-- Deputy Chairperson
3.	Prof. Kalaiselvi, MD Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Prof. Bhavani Shankar, M.S. Prof & HOD of General Surgery, MMC, Ch-3.	-- Member
6.	Prof. V. Padmavathi, M.D. I/c Director of Pathology, MMC, Ch-3.	-- Member
7.	Thiru. S. Govindasamy, BABL	-- Lawyer
8.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist
9.	Thiru. S. Ramesh Kumar, Administrative Officer, MMC, Ch-3.	-- Layperson

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

13/3/14

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Member Secretary, Ethics Committee

13/3/14

INFORMATION SHEET

- We are conducting a study on "**STUDY ON DIAGNOSTIC VALUE OF SPOT URINE PROTEINCREATININE RATIO WITH 24 HOUR URINE PROTEIN RATIO IN WOMEN WITH PREECLAMPSIA** among patients attending Kasturba Gandhi Government Hospital Chennai and for that your clinical details may be valuable to us.

- We are selecting certain patients and if you are found eligible, we may be using your clinical details in such a way so as to not affect your final report or management.

- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in this study is voluntary. Youare free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date:

STUDY ON DIAGNOSTIC VALUE OF SPOT URINE PROTEIN CREATININE RATIO WITH 24 HOUR URINE PROTEIN RATIO IN WOMEN WITH PREECLAMPSIA

PROFORMA

NAME :ADDRESS :
AGE: I.P NO:
C/O SINCE WHEN:

AGE :
OBSTETRIC HISTORY:
RISK FACTORS ASSOCIATED:
GENERAL EXAMINATION:
SYSTEMIC EXAMINATION CLINICAL EXAMINATION:
P/A:
P/S:
P/V:

Blood Pressure:The BP measured with an appropriate size cuff with the patient in an upright position after at least 10 minutes of rest.Diastolic BP was determined as the disappearance of sound or Korotkoff sound phase V.

Urine albuminuria determined by sulfasalicilic method
Urine routine analysis , urine Culture Sensitivity to exclude infection

If urine albumin > 1+ and BP >140/90 mmhg, SPOT URINE ALBUMIN CREATININE RATIO ESTIMATION DONE and then 24 HOUR URINE ALBUMINURIA CREATININE RATIO estimation done.The results of 150 patients will be analysed.

INTRODUCTION

According to the World Health Organisation hypertensive disease during pregnancy is a major cause of maternal and perinatal mortality and morbidity. Preeclampsia occurring in 3% to 8% of pregnancy is a major cause of maternal mortality, and it accounts for about 15% to 20% of iatrogenic preterm birth, Intrauterine growth retardation and perinatal mortality. In pregnancy ,preeclampsia is characterised by varying degrees of dysfunction of placenta and maternal response that shows features of systemic inflammation. Most consider hypertension and proteinuria to be the important hallmark of preeclampsia. As the severity of the proteinuria increases, there is also an equal increasing risk of maternal and fetal outcome as observed by Brown et al in 1995. So the principle objective in managing the preeclampsia is to predict proteinuria. Many of the Obstetrician depend up on the 24 hour urine collection method for estimating the proteinuria. Since collection of 24 hour urine proteinuria is a time consuming procedure and it is a cumbersome procedure for both the patient and also for the person handing the collection of urine. It is subjected to error because of inappropriate timing and incompleteness in

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
1	Mahadevi	3	4	1	1	3	1	1	2	2	2	1	1	0	0	4
2	Nagalaksh	1	5	1	1	3	3	3	3	3	3	2	4	0	4	4
3	Priya	3	5	1	1	3	2	2	4	4	4	2	5	1	2	1
4	Thilagam	3	4	1	2	2	2	2	3	3	4	2	3	2	2	2
5	Josphine	2	4	1	3	1	1	1	2	2	2	1	1	0	0	4
6	Lathamary	2	4	1	3	3	2	2	2	3	2	1	2	0	0	4
7	Rajalaksmi	3	5	1	2	3	2	2	2	2	2	1	2	0	0	4
8	Vani	3	5	1	2	2	1	1	1	0	1	1	3	0	0	4
9	Sulthana	1	5	1	1	3	1	1	1	1	1	2	2	0	3	3
10	Vasanthi	3	5	1	2	3	3	3	3	4	4	2	1	3	3	3
11	Devi	2	4	1	1	2	3	3	2	2	2	2	4	2	2	3
12	Prema	3	5	1	1	3	1	1	3	3	3	2	3	2	5	3
13	Nanthini	1	5	1	1	3	2	2	2	2	2	1	1	0	0	4
14	Barathy	3	4	1	1	2	2	3	3	3	3	2	3	7	1	0
15	Lakshmi	4	5	1	4	2	1	1	1	1	0	1	2	0	0	4
16	Suseela	1	5	1	1	2	3	3	4	4	4	2	3	2	2	1
17	Kavitha	4	5	1	3	1	1	1	1	1	1	1	2	0	0	4
18	Revathy	2	5	1	1	3	1	1	2	3	2	1	3	0	0	4
19	Sivagami	2	5	1	3	3	2	2	2	3	3	2	2	2	2	2
20	Vani	3	5	1	2	2	1	1	1	1	1	1	3	0	0	4
21	Amudha	2	4	1	2	2	1	1	1	0	1	1	3	0	0	4

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
22	Gayathri	1	4	1	1	2	1	1	1	2	1	1	3	0	0	4
23	Balkees	3	3	1	3	2	1	1	1	1	1	1	2	0	0	4
24	Sudha	3	5	1	1	3	3	3	2	2	2	1	1	0	0	4
25	Thulasi	3	4	1	1	3	1	1	1	1	1	1	1	0	4	4
26	Murugama	3	5	1	1	3	1	1	2	2	1	1	1	0	0	4
27	Lalitha	3	5	1	2	3	2	2	2	2	2	1	1	0	0	4
28	Vidhya	1	5	1	1	2	1	1	2	2	2	1	3	0	0	4
29	Parimala	3	5	1	1	3	1	1	2	2	2	1	3	0	2	3
30	Jeyanthi	2	4	1	3	3	2	2	2	2	2	1	1	0	0	4
31	Malar	2	5	1	2	2	3	3	3	3	3	2	4	1	2	2
32	Vijaya	24	5	1	1	3	1	1	1	1	0	1	2	0	0	4
33	Rebecca	1	5	1	1	3	1	1	1	1	0	1	2	0	0	4
34	Thamarai	3	5	1	3	3	3	3	3	3	3	2	2	8	3	3
35	Latha	3	5	1	2	2	2	2	1	0	1	1	3	0	0	4
36	Kumudha	3	5	1	1	3	1	1	2	2	2	1	1	0	0	4
37	Ayesha	2	5	1	1	3	1	1	2	3	3	1	1	5	4	4
38	Suganya	2	5	1	1	2	1	1	1	1	1	1	3	0	0	4
39	Kamatchi	3	5	1	2	3	1	1	1	0	1	1	3	0	0	4
40	Abirami	2	5	1	1	3	2	2	2	1	2	1	3	0	0	4
41	Selvi	1	5	1	1	3	1	1	2	2	2	1	3	0	4	4
42	Vijaya	2	4	1	2	2	2	2	1	1	1	1	2	0	0	4
43	Sarasu	4	5	1	1	1	2	2	3	3	3	2	3	0	1	0
44	Rani	3	5	1	1	1	1	1	2	2	2	1	3	0	5	3

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
45	Megalai	3	5	2	2	3	2	2	2	3	3	1	1	0	4	4
46	Suganya	4	5	1	2	2	1	1	1	1	1	1	2	0	0	4
47	Anandhi	1	5	1	1	3	1	1	1	1	1	1	3	0	4	4
48	Sasikala	4	5	1	4	2	2	2	2	3	2	1	2	0	0	4
49	Raji	2	5	1	1	3	1	1	2	2	2	1	2	0	5	4
50	Priya	4	5	1	3	1	1	1	2	2	2	1	2	0	3	4
51	Myhili	2	5	1	1	2	1	1	1	1	0	1	3	0	0	4
52	Nasreen	1	5	1	1	3	2	2	3	3	3	2	4	1	2	3
53	Rathi	3	5	1	3	3	1	1	1	1	1	1	1	0	0	4
54	Shalini		5	1	2	2	2	2	1	1	1	1	2	0	0	4
55	Karpagam	3	5	1	1	3	1	1	2	2	2	1	1	0	0	4
56	Lakshmi	2	5	1	1	2	2	2	2	2	2	1	3	0	2	3
57	Shayina	3	5	1	2	3	3	3	3	2	2	2	1	2	3	3
58	Thangam	3	5	1	1	2	1	1	1	1	0	1	3	0	0	4
59	Nirmala	3	5	1	2	1	1	1	1	0	1	1	2	0	0	4
60	Padma	4	5	1	2	3	2	2	2	2	2	1	1	0	4	4
61	Devagi	1	5	1	1	3	2	2	2	2	2	1	2	0	0	4
62	Jayasree	2	5	1	1	3	2	2	1	1	1	1	1	0	4	4
63	Soroja	3	4	1	1	1	3	3	4	4	4	2	3	8	0	2
64	Valli	2	5	1	3	2	2	2	2	2	2	1	2	0	5	5
65	Rani	2	5	1	2	2	2	2	2	2	2	1	3	2	2	3
65	Gomathi	2	5	1	1	3	1	1	2	2	2	1	3	0	0	4
66	Ponni	3	5	1	1	2	3	3	3	3	3	2	3	2	2	3

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
67	Nandhini	3	5	1	3	3	2	2	2	3	2	1	1	3	0	4
68	Deepa	3	5	1	1	3	2	2	3	2	2	1	2	8	2	3
69	Jothi	4	5	1	3	3	2	2	1	1	1	1	1	0	0	4
70	Kasthuri	2	4	1	1	3	2	2	2	2	4	1	2	0	0	3
71	Manjula	3	5	1	1	2	1	1	1	0	1	1	3	0	0	4
72	Anusiya	4	5	2	1	2	1	1	1	1	1	1	3	0	0	4
73	Meena	1	5	1	1	3	2	2	2	3	2	1	1	0	0	4
74	Saroja	1	5	1	1	3	2	2	2	3	2	1	1	0	0	4
75	Sujatha	1	5	1	2	1	1	1	2	2	2	1	2	0	3	3
76	Abirami	2	5	1	3	2	2	2	1	1	1	1	3	0	0	4
77	Kumari	4	5	1	3	3	3	3	3	4	4	2	2	2	4	4
78	Sajitha	3	5	1	1	1	3	3	3	3	3	2	3	4	1	1
79	Priya	3	5	1	2	1	1	1	1	1	0	1	2	0	0	4
80	Deepa	2	5	1	1	2	2	2	2	2	1	1	3	0	0	4
81	Karthiga	3	5	1	2	3	2	2	3	3	3	2	4	5	5	3
82	Ponmalar	1	5	1	1	2	1	1	2	2	2	1	3	0	0	4
83	Suganya	3	5	1	2	2	2	2	2	2	2	1	2	0	3	3
84	Eshwari	4	4	1	3	2	1	1	1	1	0	1	2	0	0	4
85	Kanimozhi	3	5	1	1	2	2	2	2	2	1	1	3	6	3	3
86	Poomani	2	5	1	1	3	1	1	2	2	1	1	1	0	0	4
87	Amaravaty	3	4	1	3	2	1	1	1	1	0	1	2	0	3	3
88	Tamilarsi	4	5	1	4	3	2	2	1	1	1	1	1	0	0	4
89	Aiswarya	3	5	1	1	3	2	2	1	1	1	1	3	0	2	1

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
90	Ezhil	3	5	1	1	3	2	2	3	4	4	2	4	1	1	0
91	Thirumani	3	5	1	1	2	1	1	1	1	0	1	3	0	0	4
92	Malathy	2	5	1	1	3	2	2	3	4	4	2	2	4	4	4
93	Puspa	2	5	1	2	1	1	1	2	2	2	1	3	0	2	1
94	Savithri	3	5	1	2	3	2	2	2	1	1	1	2	0	0	4
95	Indirani	2	5	1	1	2	1	1	1	1	0	1	3	0	0	4
96	Nitya	2	5	1	2	3	1	1	1	1	1	1	1	0	0	4
97	Subasini	3	5	1	1	2	2	2	2	2	2	1	3	2	3	2
98	Revathy	2	5	1	1	3	1	1	2	2	2	1	3	8	3	2
99	Savitri	3	5	1	1	2	3	3	3	3	3	1	3	2	2	2
100	Gomaty	4	4	1	3	3	1	1	1	0	1	1	2	0	4	4
101	Prema	1	5	2	1	3	2	3	3	3	3	2	4	8	2	3
102	Lalitha	2	5	1	3	2	2	2	2	2	2	1	2	0	0	4
103	Devi	3	5	1	3	2	2	2	2	2	2	1	1	0	2	4
104	Radha	2	4	1	1	2	3	3	4	4	3	2	4	8	2	3
105	Sarala	3	4	1	2	3	2	2	2	2	2	1	2	2	0	4
106	Neela	4	5	1	2	2	3	3	3	3	3	2	2	2	2	3
107	Mohana	4	5	1	2	1	1	1	2	3	3	1	3	0	2	3
108	Vijaya	3	5	1	1	3	2	2	2	4	4	2	4	4	4	4
109	Banu	1	5	1	1	3	1	1	2	2	2	1	2	0	0	4
110	Faritha	2	5	1	2	3	2	2	2	2	2	1	1	6	0	4
111	Princy	3	5	1	1	2	2	2	2	2	2	1	3	0	0	4
112	Umarani	2	5	1	3	3	2	2	2	2	2	1	1	0	2	4

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
113	Ayesha	2	5	1	1	3	1	1	2	1	2	1	1	0	2	4
114	Janani	2	4	2	2	3	2	2	2	2	2	1	2	2	3	3
115	Kavitha	2	4	1	1	3	1	1	1	1	1	1	1	0	4	4
116	Eswari	2	5	1	2	2	1	1	2	1	1	1	2	0	3	3
117	Puspha	4	5	1	2	1	1	11	1	1	1	1	1	0	0	4
118	Suganya	2	5	1	1	2	1	1	2	2	1	1	1	0	5	3
119	Malar	3	5	1	1	1	2	2	3	4	4	2	3	7	2	1
120	Manjula	2	5	1	2	3	1	1	1	0	1	1	2	2	4	4
121	Devi	2	5	1	1	3	1	1	3	4	4	2	2	2	4	4
122	Usharani	4	5	1	3	3	2	2	2	2	2	1	3	0	5	4
123	Chandra	2	5	1	1	2	2	2	2	2	2	1	3	0	2	4
124	Kamala	2	4	2	2	2	1	1	1	1	0	1	2	0	0	3
125	Prema	3	5	1	1	3	2	2	3	3	3	2	4	6	4	4
126	Ganga	4	5	1	1	3	3	3	3	4	3	2	4	5	3	3
127	Lalitha	3	4	1	1	1	3	3	4	4	4	2	3	8	2	1
128	Malrkodi	2	4	1	1	2	1	1	1	0	1	1	3	0	0	4
129	Sarada	3	5	1	3	1	2	2	2	2	2	1	2	0	0	4
130	Megalai	1	4	1	1	3	1	1	1	1	1	1	2	0	4	4
131	Kamala	2	5	1	2	2	3	3	4	4	4	2	4	1	1	0
132	Celamal	2	4	1	1	2	2	2	1	1	0	1	2	0	4	4
133	Hemalata	2	4	1	1	3	1	1	1	0	1	1	1	4	0	4
134	Jayasree	4	5	2	4	3	2	2	2	1	1	1	1	0	4	4
135	Hamidha	2	5	1	2	3	1	1	1	1	1	1	1	0	3	4

S. NO	NAME	AGE	SES	B/I	OBST.SC	GA	SYS.BP	DIA.BP	U/A	SPOT P/C	24 HUP	TREAT	M/O DELI	MAT.OC	FET.OC	B.WT
136	Thilagam	2	5	1	1	3	1	1	1	1	1	1	1	0	0	4
137	Jamila	2	5	1	3	2	2	2	2	2	1	1	1	0	0	4
138	Shanti	2	4	1	2	2	2	2	3	4	3	2	3	2	2	3
139	Buvana	2	5	1	2	3	2	2	2	2	2	1	1	0	5	4
140	Nitya	2	5	1	1	2	2	2	2	2	2	1	3	4	2	3
141	Sangeeta	2	4	1	2	3	1	1	1	1	0	1	2	0	5	3
142	Malar	2	5	1	1	3	2	2	1	1	0	1	1	0	5	3
143	Aruna	2	5	1	1	3	1	1	2	2	2	1	3	0	2	3
144	Fathima	2	5	1	1	1	2	2	2	2	2	1	1	0	2	2
145	Mary	2	5	1	1	2	2	2	2	2	2	1	1	0	0	4
146	Chandra	4	5	1	3	2	1	1	2	2	2	1	1	0	2	3
147	Janaki	3	4	2	1	2	2	2	2	2	2	1	1	0	2	3
148	Mumtaj	3	5	1	3	1	1	1	1	0	1	1	1	0	0	4
149	Kalavani	2	5	1	1	2	3	3	3	4	4	2	4	5	0	4
150	Priya	3	5	1	3	1	1	3	3	3	3	2	3	2	5	3

Column 1 Age distribution.

1 <20 years.

2 20 to 25 years.

3 26 to30 years.

4 >3o years.

Column 2 - Socioeconomic status.

1 -Class 1.

2 Class 2.

3 Class 3.

4 Class 4.

5 Class 5.

Column 3Booked and immunised.

1 Booked in kgh.

2 Booked elsewhere.

3 Unbooked.

Column 4 obstetric code.

1 - primi.

2 -gravida 2.

3 gravida 3.

4 Gravida 4.

Column -5 Gestational age.

1 -20 to 30 weeks.

2 -31 to 34 weeks.

3 ->35 weeks.

Column - 6 Systolic blood pressure.

1 -140 to 149 mmhg.

2 150 to 159 mmhg.

3 >160 mmhg.

Column -7 Diastolic blood pressure.

1 90 to 99 mmhg.

2 100 to 109 mmhg.

3 >110 mmhg.

Column 8 Urine albumin by dip sticks method.

1 -1+.

2 -2+.

3 -3+.

4 -4+.

Column –9 Spot p/c ratios.

0 -<.3.

1 - .3 to .9

2 - 1 to 1.5

3 - 2 to 2.5

4 ->2.5

Column -10 - 24 hour urine protein creatinine ratio.

0 -<300mg/day.

1 -300 to 1000mg/day.

2 1001 to 2000mg/day.

3 -2001 to 3 000mg/day.

4 ->3001mg/day.

Column - 11 Management.

1 -anti hypertensive.

2 -antihypertensives and mgso4.

Column -12 Mode of delivery .

1 -spontaneous.

2 -arm/oxytocin.

3 Pge2/oxytocin.

4 Lscs.

Column -13 Maternal outcome.

1 -Abruption.

2 Elevated liver enzymes.

3 -Hellp.

4 –elevated renal function test.

5 –pulmonary edema.

6 – Eclampsia.

7 –Systemic hypertension.

8 –Neurological symptoms.

Column 14 -Fetal outcome.

1 – Iud.

2 - Preterm.

3 –Iugr.

4 -Fetal distress.

5 -Small for date.

Column -15 Birth weight

1 -<1.5 kg.

2 -1.6 to 2kg.

3 -2 to 2.5 kg.

4 >2.5 kg.